JUN 25 1928

The New England JOURNAL of MEDICINE

FORMERLY THE BOSTON MEDICAL AND SURGICAL JOURNAL

OFFICIAL ORGAN OF
THE MASSACHUSETTS MEDICAL SOCIETY
THE NEW HAMPSHIRE MEDICAL SOCIETY—THE VERMONT STATE MEDICAL SOCIETY
THE NEW ENGLAND SURGICAL SOCIETY—THE BOSTON SURGICAL SOCIETY, INC.
THE NEW ENGLAND PEDIATRIC SOCIETY AND
THE NEW HAMPSHIRE SURGICAL CLUB

OWNED AND PUBLISHED BY THE MASSACHUSETTS MEDICAL SOCIETY

Volume 198, Number 18
$6 per Annum, 25c per Copy

JUNE 21, 1928

Published Weekly in Boston
at 126 Massachusetts Avenue

CONTENTS

THE NEW ENGLAND SURGICAL SOCIETY

Sarcoma of the Uterus. *Richard H. Miller* and *Horatio Rogers* 927

The Influence of Parathormone on Bone Regeneration. *Jacob Fine* and *Samuel Brown* 932

Psychopathology and Treatment of the Psychoneuroses. *William B. Terhune* 937

Minim Method for Analysis of Gastric Content. *Moses Einhorn* 941

Differential Diagnosis in Pulmonary Diseases. *Mark H. Joress* 943

The Interpretation of Apical Râles in Pulmonary Tuberculosis. *Randall Clifford* 948

THE MASSACHUSETTS TUBERCULOSIS LEAGUE

Fundamentals in Tuberculosis Work with Respect to Voluntary Unofficial Associations. *George J. Nelbach* 952

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Case 14181—Edema of the Feet. *Richard C. Cabot.* Case 14182—Neglected Hematuria with Fatal Result. *Harvard H. Crabtree* 956

EDITORIALS

Coöperation 963
The Filterable Viruses 964
Some Deficiencies in Dealing with Tuberculosis. 964
This Week's Issue 965
The Annual Meeting of the American Medical Association 966
The Meeting of the Harvard Medical School Alumni in Minneapolis 967

(*Contents continued on next page*)

Bethea's Clinical Medicine

Just Ready

There is a field of internal medicine, Dr. Bethea says, that in all his years as practicing physician, teacher of clinical medicine and of therapeutics, has impressed him as reaching out beyond all others for help from the "school of experience." The field is that of the general practitioner away from the large medical centers. Not having at hand the extensive equipment of the hospital these physicians must diagnose and treat disease in the office and in the home with scant, if any, equipment other than their senses and experience. In his lectures to students, Dr. Bethea has taught with this group in mind; and now, in equally cogent style, has incorporated his teaching in this volume. Theory holds but little sway in this book. Experience—practice dominate.

The unusual clarity with which the subject is outlined is due undoubtedly to Dr. Bethea's simple approach. Always his aim is to simplify, to interest, to carry the reader on to a complete and thorough understanding of the disease.

Dr. Bethea clearly explains the diagnostic procedures necessary to reveal the disease and to bring sharply into focus all the factors which help to individualize it. In conditions where confusion might arise, he takes up the differential diagnosis. Symptoms, because of the particularly important part they play in diagnosis, are described at great length.

The presentation of treatment—*bedside treatment*—is a feature which of itself, should make the book the thumb-worn volume in every physician's library. Here is a simple discussion which presents the treatment of disease from every important angle, including such general considerations as the patient's quarters, surroundings, care, etc. Symptomatic treatment is described in every case, giving Dr. Bethea's own prescriptions. Diet is discussed in a clear and yet scientific way.

Simplicity and readableness add much to the book. As you read it you feel that here is not a dry discourse on disease, but a fresh exposition of a successful physician's methods of diagnosis and treatment.

Octavo volume of 700 pages, illustrated. By OSCAR W. BETHEA, M.D., Professor of Clinical Therapeutics, Tulane University School of Medicine, New Orleans. Cloth, $7.50 net.

W. B. SAUNDERS COMPANY — **Philadelphia and London**

Entered at the Postoffice at Boston as Second Class Matter

CONTENTS

(Continued)

BOOK REVIEWS

An Elementary Text-Book of General Microbiology. Ward Giltner 970
Filterable Viruses 970
The Ultra-Violet Rays. Arnold Lorand 970

BOSTON MEDICAL LIBRARY

Extension Courses for Rural Practitioners 968

CORRESPONDENCE

Official Actions by the Board of Registration in Medicine. Frank M. Vaughan 970

MISCELLANY

New York Medical Center 969
Norfolk County Health Association Study 969

NOTICE 970

REPORT OF MEETING 970

SOCIETY MEETINGS 970

The Massachusetts Medical Society

The next Annual Meeting of the Massachusetts Medical Society will be held in Boston, June 11 and 12, 1929, "unless otherwise ordered by the Council."

OFFICERS ELECTED BY THE COUNCIL, JUNE 5, 1928

PRESIDENT: JOHN M. BIRNIE, 14 Chestnut St., Springfield.
VICE-PRESIDENT: PEER P. JOHNSON, 163 Cabot St., Beverly.
SECRETARY: WALTER L. BURRAGE, 182 Walnut St., Brookline.
TREASURER: ARTHUR K. STONE, Auburn St., Framingham Center.

STANDING COMMITTEES FOR 1928-1929

COMMITTEE OF ARRANGEMENTS
James Hitchcock, E. P. Hayden, H. Q. Gallupe, T. H. Lanman, F. H. Colby.

COMMITTEE ON PUBLICATIONS
Homer Gage, John W. Bartol, R. I. Lee, R. B. Osgood, E. W. Taylor.

COMMITTEE ON MEMBERSHIP AND FINANCE
D. N. Blakely, Algernon Coolidge, Samuel Crowell, Gilman Osgood, Homer Gage.

COMMITTEE ON ETHICS AND DISCIPLINE
David Cheever, W. D. Ruston, S. F. McKeen, Kendall Emerson, A. C. Smith.

COMMITTEE ON MEDICAL EDUCATION AND MEDICAL DIPLOMAS
P. P. Johnson, A. G. Howard, R. L. DeNormandie, H. P. Stevens, C. H. Lawrence.

COMMITTEE ON STATE AND NATIONAL LEGISLATION
J. M. Birnie, A. W. Marsh, T. J. O'Brien, F. E. Jones, Shields Warren.

COMMITTEE ON PUBLIC HEALTH
Dwight O'Hara, E. F. Cody, R. I. Lee, T. F. Kenney, F. G. Curtis.

COMMITTEE ON MALPRACTICE DEFENSE
F. G. Balch, E. D. Gardner, F. B. Sweet, R. P. Watkins, A. W. Allen.

OFFICERS OF THE SECTIONS FOR 1929

SECTION OF MEDICINE
Chairman, Dr. Lester C. Miller, Worcester; *Secretary*, Dr. Roger W. Schofield, Worcester.

SECTION OF SURGERY
Chairman, Dr. Philemon E. Truesdale, Fall River; *Secretary*, Dr. L. S. McKittrick, Boston.

SECTION OF TUBERCULOSIS
Chairman, Dr. John B. Hawes, 2nd, Boston; *Secretary*, Dr. Walter A. Griffin, Sharon.

SECTION OF PEDIATRICS
Chairman, Dr. C. A. Sparrow, Worcester; *Secretary*, Dr. Joseph Garland, Boston.

SECTION OF OBSTETRICS AND GYNECOLOGY
Chairman, Dr. Frederick L. Good, Boston; *Secretary*, Dr. Louis E. Phaneuf, Boston.

SECTION OF RADIOLOGY AND PHYSIOTHERAPY
Chairman, Dr. William D. McFee, Haverhill; *Secretary*, Dr. P. F. Butler, Boston.

OFFICERS OF THE DISTRICT MEDICAL SOCIETIES

ELECTED BY THE DISTRICT MEDICAL SOCIETIES BETWEEN APRIL 15 AND MAY 15, 1928

BARNSTABLE—*President*, S. M. Beale, Jr., Sandwich; *Vice-President*, H. D. Handy, Harwich; *Secretary*, J. L. Chute, Osterville; *Treasurer*, H. B. Hart, Yarmouthport; *Librarian*, E. E. Hawes, Hyannis.

BERKSHIRE—*President*, C. H. Richardson, Pittsfield; *Vice-President*, M. S. Eisner, Pittsfield; *Secretary*, H. J. Downey, Pittsfield; *Treasurer*, C. T. Leslie, Pittsfield.

BRISTOL NORTH—*President*, F. H. Dunbar, Mansfield; *Vice-President*, T. J. Robinson, Taunton; *Secretary*, J. L. Murphy, Taunton; *Treasurer*, J. V. Chatigny, Taunton.

BRISTOL SOUTH—*President*, J. G. Hathaway, New Bedford; *Vice-President*, G. L. Richards, Fall River; *Secretary and Treasurer*, G. E. Borden, Fall River.

ESSEX NORTH—*President*, G. E. Kurth, Lawrence; *Vice-President*, R. L. Toppan, Newburyport; *Secretary and Treasurer*, J. Forrest Burnham, Lawrence.

ESSEX SOUTH—*President*, O. S. Pettingill, Middleton; *Vice-President*, C. L. Curtis, Salem; *Secretary*, R. E. Stone, Beverly; *Treasurer*, Andrew Nichols, 3d, Danvers; *Librarian*, C. M. Cobb, Lynn.

FRANKLIN—*President*, A. H. Ellis, Greenfield; *Vice-President*, W. H. Clark, Greenfield; *Secretary and Treasurer*, Charles Moline, Sunderland.

HAMPDEN—*President*, E. P. Bagg, Jr., Holyoke; *Vice-President*, Dudley Carleton, Springfield; *Secretary and Treasurer*, H. L. Smith, Springfield.

HAMPSHIRE—*President*, F. E. O'Brien, Haydenville; *Vice-President*, L. B. Pond, Easthampton; *Secretary and Treasurer*, L. O. Whitman, Northampton; *Librarian*, F. H. Smith, Hadley.

MIDDLESEX EAST—*President*, D. T. Buzzell, Wilmington; *Vice-President*, F. T. Woodbury, Wakefield; *Secretary*, A. R. Cunningham, Winchester; *Treasurer*, Richard Dutton, Wakefield; *Librarian*, J. M. Wilcox, Woburn.

MIDDLESEX NORTH—*President*, J. H. Lambert, Lowell; *Vice-President*, A. G. Scoboria, Chelmsford; *Secretary*, T. A. Stamas, Lowell; *Treasurer*, M. D. Bryant, Lowell; *Librarian*, P. J. Meehan, Lowell.

MIDDLESEX SOUTH—*President*, A. W. Dudley, Cambridge; *Vice-President*, Fresenius Van Nüys, Weston; *Secretary*, S. M. Biddle, Cambridge; *Treasurer*, Edward Mellus, Newton.

NORFOLK—*President*, Victor Safford, Jamaica Plain; *Vice-President*, S. F. McKeen, Brookline; *Secretary*, F. S. Cruickshank, Dorchester; *Treasurer*, G. W. Kaan, Reading.

NORFOLK SOUTH—*President*, D. A. Bruce, Atlantic; *Vice-President*, G. M. Sheahan, Quincy; *Secretary, Treasurer and Librarian*, N. R. Pillsbury.

PLYMOUTH—*President*, T. H. McCarthy, Brockton; *Vice-President*, F. W. Murdock, Brockton; *Secretary and Treasurer*, L. B. Packard, Brockton; *Librarian*, J. H. Weller, State Farm.

SUFFOLK—*President*, Lincoln Davis, Boston; *Vice-President*, W. H. Robey, Boston; *Secretary*, A. H. Crosbie, Boston; *Treasurer*, John Rock, Boston.

WORCESTER—*President*, P. H. Cook, Worcester; *Vice-President*, A. M. Shattuck, Worcester; *Secretary*, C. A. Sparrow, Worcester; *Treasurer*, G. O. Ward, Worcester; *Librarian*, A. C. Getchell, Worcester.

WORCESTER NORTH—*President*, J. H. Kearney, Fitchburg; *Vice-President*, C. S. Brigham, Leominster; *Secretary*, C. H. Jennings, Fitchburg; *Treasurer*, F. H. Thompson, Jr., Fitchburg.

The New England
Journal of Medicine

VOLUME 198 · JUNE 21, 1928 · NUMBER 18

NEW ENGLAND SURGICAL SOCIETY

SARCOMA OF THE UTERUS*

BY RICHARD H. MILLER, M.D., F.A.C.S., AND HORATIO ROGERS, M.D.†

IT was in 1860 that Virchow called the attention of the medical profession to the occurrence and importance of sarcoma of the uterus. Since that time there have appeared numerous reports both of isolated cases and collected series, but an occasional refreshing of our knowledge of this rather unusual condition is not without value.

Of course, most cases of sarcoma of the uterus are discovered during or after an operation for fibroid tumor, and therefore the frequency of occurrence is usually computed in the percentage of cases found in the pathological examination of a series of fibroids. Figures vary, but the majority of observers estimate the number of sarcomata as being from 0.5% to 2.0% of the myomata. Miller, from 9750 cases, reported sarcomata in 1.9%, Noble 1.4% of 2274 cases, Kelly and Cullen 1.2% of 1400 cases, and Deaver and Pfeiffer 1.2% of 345 cases (Maroney[1]). Masson[2], at the Mayo Clinic, reported 44 sarcomata to 4322 fibroids, or 1%. Vogt[3] found only 0.6% in 1216 cases, and from the literature collected 72,116 cases with sarcoma in 0.4%. On the other hand, Imhäuser[4] estimates that the figure is as high as 6.%. Reel and Charlton[5], reporting from the Pathological Department of the Ohio State University in 1923, show a higher figure, finding 11 cases out of 288. In a hitherto unpublished study one of us, in investigating the pathological reports of 5514 fibroid specimens, found 14 cases of sarcoma alone and 30 cases of sarcoma plus myoma—a total of 44, or 0.8%. It must be borne in mind that the reaction of the individual pathologist has a great bearing on his interpretation of a given series; for instance, Warner, in a careful study of 100 consecutive cases, finds two definite sarcomatous and 5 others "very cellular, and well on the border line of this malignant complication." Another pathologist might interpret the figure for sarcomata, in this series, as higher than 2%. However, we can accept 1% as a fairly representative figure from the literature.

The majority of sarcomata are in the fundus—about 12% occur in the cervix. Ewing[7] classifies them as:

1. Circumscribed.
2. Diffuse or infiltrating.
3. Polypoid (of body or cervix).
4. Extra-uterine, single or multiple myosarcomata.
5. Secondary sarcoma in myoma.

He further describes the "mucosal" type—that which develops in the sub-mucous connective tissue. This type may form pedunculated masses which protrude from the cervix into the vagina, and recur after removal by curettage. Graves[21] says "Where this type of sarcoma springs from the mucosa of the cervix the vagina becomes filled with a mass of grape-like polyps, which eventually protrude through the cervix into the outer world. The tumor under these conditions is commonly called grape-mole or sarcoma botryoides."

Reel and Charlton[5] in 11 cases found 9 occurring in the body and two arising from the submucous stroma—the endometrial type. Vogt[3] found in 30 cases 28 arising from the body and two from the cervix. It may fairly be said that most cases occur in the fundus and are mistaken for myomata.

The question as to whether sarcoma arises from myoma is an interesting and important one. Ewing[7] says that those occurring in the wall of the uterus are myogenic in origin and may or may not be secondary to fibroids. Evans[8] states that the sarcoma may, but usually does not, develop on or in a fibroid—it usually arises from the ordinary normal muscle cells. Warner's[6] 2 cases of sarcoma were in a series of 100 fibroids, while Reel and Charlton[5] found 8.6% of sarcomatous changes in pre-existing fibroids. Vogt's[3] 30 cases of sarcoma are interesting in that only 8 developed on pre-existing myoma—in other words, 73% occurred primarily. The figures of Frankl[9] are as follows:

Total cases of Sarcoma—38.
Associated with pre-existing myoma—17.
Probably associated with pre-existing myoma—5.
Not associated with pre-existing myoma—15.
Doubtful—1.

*Read at the Annual Meeting in Manchester, N. H., October 1, 1927.

†For record and address of author see "This Week's Issue," page 965.

The answer to this question, then, is that some sarcomata develop in myomata and some do not; accurate figures are hard to obtain. As Frankl says, there may exist in one uterus (1) myoma and sarcoma independently, (2) myoma invaded by sarcoma, and (3) sarcoma developing in a myoma.

The most commonly mentioned histological types are the spindle-cell, giant-cell, and small-round cell—the last being considered the most malignant. The ordinary microscopic characteristics of the uterine sarcomata, as observed by many authors, are (1) increase in size of cells, (2) shorter and plumper cells with oval or vesicular nuclei, (3) inequality in size and irregularity in shape and arrangement, (4) lack of differentiation, (5) unequal and deep staining of nuclei, (6) large cells with occasional multiple nuclei, (7) mitotic figures, (8) decrease or absence of stroma, and (9) thinness or absence of vessel walls (Evans[8]). Ewing, and Proper and Simpson, accept the giant-cell as evidence of a rather high degree of malignancy, but Evans and Masson do not agree. Evans[8], in a very valuable piece of work, grouped the sarcomata according to malignancy by counting the mitotic figures in 1 cu. mm. of tumor. His groups are as follows:

I 2200—12000 per cu. mm.
II 200— 800 " " "
III 0—few

He classed in this way and followed 72 cases, with these observations:

Group I —13 cases—11 died of recurrence—two living.
Group II —9 who were traced are well.
Group III—all who could be found were well.

He considers Group I highly malignant, Group II border-line—perhaps a stage of metamorphosis, and Group III premalignant. All are agreed that distant metastases are rare, and the extension is rather local and abdominal, though occasionally, especially in the round cell type, there may be metastases in the lungs.

Sarcoma and carcinoma may occur together in the same uterus, but this combination is rare; a recent case has been reported by Jaffe[10], and one of sarcoma and adenoma by Froeschmann[11]. Also of academic interest in this connection are the occasional mixed tumors of the uterus. Such have recently been reported by Perlstein[12], Wiener[13], and Petersen[14], and the last named has collected 50 cases from the literature. These tumors are analogous to the teratoid growths of the ovary, and are due to the displacement of embryonal rests along the Wolffian duct, which in the female becomes Gartner's duct. The tumors almost always contain sarcomatous tissue, and often cartilage, smooth muscles, striated muscle, fat, and less often bone and endothelium. There have also been reported 31 cases of lipoma of the uterus[14], and 43 cases of endothelioma[15].

The cases of sarcoma present no characteristic signs or symptoms. They may give the picture of myoma, cervical polyp, or unexplained uterine bleeding. Suggestive facts, however, are that they grow much more rapidly than ordinary fibroid tumors, are more invasive, and tend to recur.

As to treatment, radical surgery offers the most chance of successful cure though European authors are enthusiastic about radiation. Beclere[16], quoting Seitz and Wintz, reports 18 cases, of whom 14 were cured by radiotherapy; Imhauser also advises large doses of X-ray. We would recommend that in every hysterectomy for fibroid or unusual tumor, the specimen should be carefully examined at the time of operation, and if any evidence of sarcoma be detected, the dissection should be made more complete, with removal of the cervix.

In regard to the gross appearance, the polypoid type or "grape sarcoma" should easily be recognized as malignant at operation, as should diffuse sarcoma of the uterine mucosa. The recognition of malignant changes in myomata, however, is difficult on gross examination, and sometimes even on microscopic as has been pointed out above. The gross section of a benign undegenerated myoma has a characteristic apperance and feel. Of the various non-malignant forms of degeneration, some should cause no confusion, such as calcareous, hyaline, edematous, myxomatous, cystic, and fatty. It would be easy to mistake for malignancy very rapidly growing myomata, certain appearances of necrosis, or so-called red degeneration. However, if the cut section of a myoma presents a soft grayish-pink or yellowish appearance, is friable, and no frozen section is available, it would probably be safer to treat it as malignant until microscopic examination proves it benign.

CASES AT THE MASSACHUSETTS GENERAL HOSPITAL

From 1876 to 1926 there have been at the Massachusetts General Hospital 2043 cases of fibroid tumor, most of them being operated on. The figures up to 1897 are not accurate, but are approximately correct—at least enough so for the purposes of this study. In the same period there have been 25 cases of sarcoma—1.2%. The average age of these was 46—the youngest 28 and the oldest 64.

The location of the growth was as follows:—

Fundus	20	80%
Cervix	4	16%
Fundus and cervix	1	4%

The tumors were examined by three different pathologists, and the microscopic findings are for this reason a little hard to correlate—they are as follows:—

Spindle cell	12	48%
Myxo sarcoma	2	8%
Round cell	2	8%
Round and spindle cell	1	4%
Other types	8	32%

Of the cases 7, or 28% had local metastases, or extension of the growth, outside the uterus. The parametrial tissues, great omentum, and peritoneum of the small bowel, were the structures involved. None had distant metastases. The reports were difficult of interpretation, but in 9, or 36% there were concomitant fibroids, and it is assumed that the sarcoma developed in these; on the other hand, in 63% there was no definite report of myoma. Mitosis was reported present in but 4 cases. The autopsy protocol of one interesting case is here given:—

Autopsy No. 2781

ANATOMICAL DIAGNOSIS

Leiomyosarcoma of Uterus with Metastases in Retroperitoneal Tissue and in Peritoneum.
Anemia
Slight Hydronephrosis, rt.
Oedema of lower extremities
Operative wound

(From the Notes)

The tubes and ovaries are not found.

Situated retroperitoneally are 5 large roundish masses of new growth tissue which protrude markedly into the peritoneal cavity pressing the intestines which skirt over them markedly forward. Tissues necrotic and infiltrated with large amounts of fluid. Peritoneal cavity contains beside new growth considerable dirty brownish red fluid. Upper portion of uterus present, walls very thin and cavity rather large. Cervix and vagina not remarkable, but posterior wall of the uterus rests over and is closely applied to one of the masses of new growth tissue mentioned which completely fills the pelvic cavity. Uterine wall separates off easily from this mass. Omentum involved in this new growth and in several instances the wall of the small intestine shows smaller and larger pedunculated masses of new growth tissue similar in character to that already described. Microscopic examination: sections of tumor in several situations including uterus and intestines show that the tumor tissue consists of large spindle shaped cells with long polar processes arranged parallel to one another in bundles and with only a little connective tissue stroma in places. A few blood vessels traverse the tumor tissue.

The symptoms chiefly complained of were, as above inferred, those which might well have been caused by fibroid tumor or carcinoma. They were:—

Bleeding	17	68%
Pain	13	52%
Abdominal tumor	10	40%
Loss of weight	3	12%
Mass in vagina	2	8%
Trouble with micturition	1	4%

No definite attempt was made to follow up these cases, but the end results, as far as obtained, are given below:—

Died of the growth	5	20%
Died of the operation	3	12%
Well 1 yr. after operation	3	12%
Well 6 mos. after operation	1	4%
Well 6 yrs. after operation	1	4%
Not traced	12	48%

All the above figures from our cases bear out in a general way the reports from other clinics:—that approximately 1% of alleged fibroid tumors will turn out to be sarcomatous; that the malignancy of these varies from very slight to very great; that there are no characteristic symptoms; and finally that every effort should be made, at the time of operation, to detect them, so that as radical an operation as possible may be done.

Our interest in this subject was aroused by a case which was sufficiently extraordinary to warrant brief mention here.

CASE. A woman of 40, white, a cook, presented herself for the treatment of varicose veins. The past history was irrelevant, and her only complaint was from the veins. Examination revealed, beside the veins, a huge rounded smooth abdominal tumor, apparently solid, extending from the pubes to the ensiform, and larger than a full-term pregnancy. The patient had not known of its existence, and indeed was skeptical about it. She had no abdominal symptoms, and no disturbance of menstruation. The diagnosis of fibroid tumor was made, and operation performed. At the operation the tumor, which filled the whole abdomen, and to which the intestines were more or less adherent, was removed without any marked difficulty, and without leaving any visible masses of growth. The immediate convalescence after operation was uneventful. The pathological report follows:—

"The specimen consists of an enormous solid uterine tumor weighing 5730 grams. Both adnexa, somewhat distorted, can be recognized. On section, it is made up of a number of spherical tumors of varying sizes, some of which are firm and grayish white, while others are soft and show a pure white cut surface. The largest of these is for the most part necrotic and is soft and dull yellow in color. What appears to be the uterine cavity is considerably distorted and shows a smooth inner surface.

Microscopical examination of sections of this larger mass shows an undifferentiated, richly cellular stroma. The cells are very atypical without any definite arrangement and are generally elongated and of spindle shape. There are scattered areas of necrosis. It is bounded by a thin zone of uterine muscle in which there is no evidence of invasion. Sections of one of the larger nodules show a richly cellular stroma with less atypicality of the cells and some differentiation, the cells being arranged in interwoven bundles like a fibromyoma. Other nodules show the typical structure of a fibromyoma. The largest tumor must be regarded as malignant and suggests in its appearance a leio-myosarcoma and the other nodules fibroids of various degrees of cellular richness.

Diagnosis: Leio-myo-sarcoma."

Two months after this operation she began to complain of pain and stiffness in the region of the right hip. Six months after the operation she was admitted to Hospital with a spontaneous fracture of the right hip. At this time there were no palpable abdominal tumors, and X-ray of the spine, pelvis, and shoulder were negative. It was finally decided to explore the site of fracture and a brief report of the operation follows:—

"Usual lateral incision. Tensor fasciae femoris and gluteus medius muscle separated. At the lower

part of the incision there was a small protuberance, the size of a bean, projecting through the capsule of the joint. It was hard, fibrous and glistening, and unquestionably malignant. The capsule was opened and the whole neck found to be infiltrated with disease."

A small amount of the tissue was removed and the pathological report was as follows:

Several small irregular grayish-white pieces showing on microscopic examination a tumor composed of irregular spindle cells with long wavy fibrillae arranged in interlacing bundles. Many of them have long nuclei and some are multi-nuclear. There is considerable variation in the amount of their chromatin. Mitotic figures are numerous. This specimen resembles the one previously removed from the uterus.

The patient died shortly afterwards in a hospital for incurables after developing a distressing cough which was thought to be due to pulmonary metastases, though this was not proved.

Here, then, is a striking and most typical example of a uterine sarcoma of a high degree of malignancy. Had this patient been more intelligent and more observant, she would doubtless have presented herself for early operation, and a radical cure might have been obtained.

CONCLUSIONS

1. Twenty-five cases of sarcoma of the uterus are reviewed. They constitute in our series 1.4% of the number of fibroids.

2. Sarcoma of the uterus presents no pathognomonic signs or symptoms, and is usually mistaken for myoma.

3. It may, but does not necessarily, originate in a pre-existing fibroid.

4. Any rapidly growing fibroid tumor should be suspected of being sarcomatous.

5. At every operation for fibroid tumor of the uterus the specimen should be carefully examined to determine if sarcoma be present; if so, the operation should be made very radical.

REFERENCES

1 Maroney: Am. J. Obstetrics, 1916, Vol. 74, p. 445.
2 Masson: Am. J. Ob. and Gyn., 1923, V, 345.
3 Vogt: Am. J. Ob. and Gyn., V, No. 5, p. 523, May, 1923.
4 Imhäuser: Arch. f. Gyn., 1924, Vol. 123, p. 12.
5 Reel and Charlton: Am. Surg., LXXVII, p. 476, 1923.
6 Warner: Am. J. Obstetrics, 1917, Vol. 76, p. 888.
7 Ewing: Neoplastic Diseases, 1922.
8 Evans: S., G. and Ob., XXX, No. 3, p. 225, Mar., 1920.
9 Frankl (Vienna): Am. J. Ob. and Gyn., IX, No. 6, p. 745, June, 1925.
10 Jaffé: S., G. and Ob., XXXVII, 1923, p. 472.
11 Froeschmann: Ztschr. f. Geburts. u. Gyn., Vol. 81, p. 623, 1919.
12 Perlstein: S., G. and Ob., XXVIII, p. 43, 1919.
13 Wiener: Am. J. Ob. and Gyn., VIII, No. 2, p. 211, Aug., 1924.
14 Petersen: J. Lab. and Clin. Med., VIII, p. 369, 1923.
15 Murphy: S., G. and Ob., XXXVII, No. 1, p. 24, July, 1923.
16 Béclére: J. de Radiologie et Electrologie, Paris, Dec., 1920, IV, No. 12, p. 529.
17 Brady: Bull. J. H. Hosp., 1918, Vol. 29, p. 164.
18 Auvray: Bull. Soc. d'obst. et de Gyn. de Paris, 1923, XII, 280.
19 Jacquin: Gynecologie et Obstétrique, Paris, 1921, No. 3, 2-3, p. 90.
20 Kuncz u. Sacher: Arch. f. Gyn., CXXIII, p. 211, 1924.
21 Graves: Gynecology, Saunders, 1916.

DISCUSSION

DR. H. T. HUTCHINS, Boston: Every once in so often someone ought to bring us up to date on the rare things that occur in surgical pathology, and this Dr. Miller has done this afternoon. He has brought up sarcoma of the uterus which is a rare disease and of which we see very few cases, and he has brought it up from the histological and pathological standpoints; and we ought to be grateful to him for refreshing our memories on this.

I have nothing to add to the discussion except that in one way it seems another reason for doing panhysterectomy as we all know these cases cannot be diagnosed before operation. They are seldom diagnosed. They are diagnosed when there is a mass in the vagina. But as to the degeneration of a fibroid or a polyp into fibrosarcoma, we don't know. We get bleeding, but the curette doesn't give us the information we want. So that as in the case I mentioned in the paper, if we are going to do a hysterectomy for fibroids, why not do a panhysterectomy so that then this one per cent. of sarcoma is out of the way and the field is clear?

An interesting article by DR. WILLIAM LEWIS has appeared in the *Johns Hopkins Bulletin* wherein he says he is now able to determine the kind of a tumor from its vascular formation after the tumor is removed. He has injected fibroids and sarcomata and carcinomata and he finds a striking difference in the vascularization of these various tumors. It is a very interesting article which would well repay reading.

DR. D. W. PARKER, Manchester: Dr. Miller's very comprehensive paper needs very little discussion. I was surprised at his statistics of one per cent. of sarcoma. To my knowledge I have never had a single case of sarcoma of the uterus. This means that I have been either fortunate, or have been careless in not sending all of my fibroids for pathological examination.

One point it brings up is that we must not be misled by the apparent microscopic appearances in the specimen we remove. Of course, in the large hospitals the pathological examination is automatic. In our small hospitals here pathological examinations are not automatic. They have to be obtained sometimes with a little effort and difficulty, and sometimes we get a bit careless when we think a thing is obviously what it appears to be.

DR. CHARLES A. PORTER, Boston: I do not think that after performing multiple myomectomies that all of the fibroids would be automatically examined unless some one of them looked suspicious.

DR. STEPHEN RUSHMORE, Boston: This condition described by Dr. Miller is very interesting. He called attention to one per cent. of fibroids showing malignant or sarcomatous degeneration, a figure probably not too high for larger series of cases also. I think the more carefully fibroids are investigated, the higher the figures will be.

If we accept one per cent. of sarcomata in myomata, and then add two per cent. of carcinomata, we have a percentage at least as high as

the difference in the mortality between radical and conservative operations for myomata (panhysterectomy as compared with supra-vaginal hysterectomy.)

So we have here another suggestion and indication that the radical procedure is the operation of choice in myomata.

What is a sarcoma? Figures compiled years ago as to frequency are not strictly applicable today for it has been shown that a considerable number of leiomyomata are malignant. We shall find, I think, that the more frequently investigations are made, the higher the percentage of malignant tumors among myomata.

Dr. R. H. Miller, Boston (closing): I have nothing further to add of importance. There were occasional reports of other malignant tumors such as endothelioma, also large lipomata of the uterus, all of which are very rare and practically never diagnosed before operation.

THE INFLUENCE OF PARATHORMONE ON BONE REGENERATION*

BY JACOB FINE, M.D.,† AND SAMUEL BROWN, M.D.‡

IN 1907, Erdheim[1] reported experiments on rats which showed that, as a result of parathyroidectomy, the teeth lost their yellow transparent appearance and became white and opaque; moreover, that they grew longer and, due to increased brittleness, were distorted in shape. He noted also that there was a diminished and delayed deposition of calcium in the dentine, and that the enamel was decreased and thin, or quite lacking. Hohlbaum[2], and others, confirmed these results as well as Erdheim's further contention that reimplantation of the glands restored the teeth to normal. However, other observations made by Erdheim[3] (to the effect that parathyroidectomy caused (a) delay in the healing of fractures, (b) diminished or complete lack of calcification in newly developing bone, (c) softening and spontaneous fractures in the skeletal bones, and (d) shrinking of the zone of proliferating hypertrophic cartilage which became well calcified) were not substantiated either by Hohlbaum, by Jovane and Vaglio[4], who worked with dogs, or by Korenchewsky[5]. The latter makes the point that Erdheim's experiments suffer from lack of dietary control. Leopold and Reuss[6] analyzed seven of Erdheim's parathyroidectomized rats, but found no consistent change in the skeletal calcium from that of normal rats of the same age; on the other hand, the soft tissue calcium was increased in all of them.

In 1914, Erdheim[7] supplemented his original work with the observation that spontaneous rickets in rats was associated with hyperplasia and hypertrophy of the parathyroids. Ritter[8] found that in human rickets there was a hyperplasia of the parathyroids, and that, contrary to the normal appearance of these glands, the light cells exceeded the darker ones in number; but these changes were demonstrable only in severe cases. Pappenheimer and Minor[9] found a similar condition in these cases, and Doyle[10] reported, in 1925, an enlargement of the parathyroids in rachitic chickens which had latent tetany and calcium deficiency. He stated that this enlargement was characteristic enough to make this finding a method of differential diagnosis between the leg weakness of rickets and that due to other causes.

Tomeszewski[11], in 1912, investigated the parathyroids in twenty cases of pathological calcification,—e. g. that found in tuberculous processes, arteriosclerosis, and early calcifying ribs and larynx,—and in many cases discovered an increase in the number and size of the oxyphil cells. Todyo[12], in 1912, described a hyperplasia of the glands in six out of seven cases of osteomalacia, and in eight out of eleven of osteoporosis, whereas only four of twenty-four normal skeletons showed such changes. But Ritter[13] found the degree of hyperplasia variable. Meyer[14], in 1917, described a case of osteitis fibrosa cystica associated with a benign tumor made up of normal-looking parathyroid tissue. Findings similar to Meyer's were reported by Schmorl[15], Bauer[16] and Hohlbaum[17]. Recently Dr. Harvey Cushing told one of us of a case of acromegaly in which he found at autopsy a tumor of the parathyroids. Goedel[18] reported a cancer of the parathyroid associated with osteitis fibrosa cystica. On the basis of such observations extirpation of the parathyroids was practiced in a case of osteitis fibrosa reported by Mandl[19].

Glison[20] removed the external pair of glands with the adherent portion of thyroid in young dogs, and obtained an arrest of bone growth with death from cachexia in four months. Hammett[21], studying for seventy-five days the growth of the humerus in a thyroparathyroidectomized rat, found its development retarded, but this was proportionately less than the retardation of growth of the animal as a whole.

Ogawa[22] investigated the relation of the gland to the healing of fractures and reported that union was slower following removal of the glands. Dieterich[23] found that this procedure delayed callus formation, but that X-ray and histological examination proved that otherwise the process was normal in every respect.

Morel[24], in 1909, found in cats a marked retardation in the calcification of callus after parathyroidectomy, but only in young animals, not in adults. He injected a parathyroid extract subcutaneously, which, in young animals, produced bones twice the thickness of his controls, regardless of the amount of calcium in the food.

Some of the above experiments are called into question as a result of recent work by Gates and Grant[25], who demonstrated fairly conclusively that subtotal removal of the parathyroids creates a deficiency which is rapidly compensated by hypertrophy of the remaining tissue, and that the blood calcium is restored to normal in a week to ten days. Anything short of complete removal therefore is not a reliable test of underfunction of the tissue after this period of time. Furthermore, Swingle and Nicholas[26] have shown that, for example, in the cat, it is very difficult to totally remove all the parathyroid tissue on account of the widespread distribution

*These experiments were carried out at the Department of Surgical Research of the Harvard Medical School at the suggestion of Dr. E. P. Richardson. We are indebted to him and to Dr. J. C. Aub for very kind guidance and advice. The work was greatly facilitated by the kind co-operation of Dr. A. J. McLean, Austin Fellow in charge of the laboratory, to whom we wish to express our thanks.

†Recipient of Dalton Scholarship for 1926-7 from Massachusetts General Hospital.

‡For records and addresses of authors see "This Week's Issue," page 965.

of accessory glands in the cervical fascia and the thymus.

In 1924 Collip[27] isolated the active principle of the gland and thus made possible a more precise study of the effect of the hormone on the metabolism of bone.

So far we have been unable to find anything in the literature on the effect of the extract on the calcification of bone aside from scattered statements referring to its use in abnormal cases of bone metabolism.

FIG. 1

Corresponding femora of 2 dogs (young adults) (Nos. 1 and 3, respectively) showing healing of lesion (button of bone removed by trephine) 53 days after operation. Dog 3 received 490 units of parathyroid extract over a period of 40 days (half the full physiological dose determined by Collip). Dog 1 received no extract. Result: No essential difference in calcification.

Our experiments on this aspect of the subject were done on dogs. They consisted of a study of bone repair under the influence of the extract.* The injury of the bone which we first produced was a trephine opening in the femur. This was done on four adult dogs, two of which received full physiological doses of the extract.

There was no marked difference in the resulting regeneration as far as was evident from X-ray or from gross or microscopic study of the specimens. (See Figs. 1 and 2.) But we soon abandoned this procedure because of the variable factor arising from the impossibility of reapproximating the periosteum when only a button of bone is removed. Instead, we preferred to remove a rib subperiosteally and watch the effect on a perfectly splinted bone lesion in which the role of the periosteum could be more or less standardized. The dogs were kept isolated and on a milk and meat diet, so that an adequate calcium intake was provided. In each experiment two dogs as nearly alike in age and size as could be obtained were used, one of them receiving the extract for the greater part of the postoperative period. Adult and young dogs were run, using different doses of the extract and allowing varying periods of time for bone repair. Histological examination of the specimens revealed nothing not already obvious from the X-ray studies.

FIG. 2

Dogs 2 and 4 (young adults) treated in same way as Dogs 1 and 3 (see legend for Fig. 1). Dog 2 (femur on left) was the control. Dog 4 received 480 units (full physiological dose) over a period of 3 weeks following operation and died from parathormone poisoning. Dog 2 sacrificed on same day. Result: No essential difference in calcification.

The first two sets of observations were obtained on four vigorous young adult dogs, one-half the full physiological dose being used on the first set of animals. Subcutaneous injections were made every day or every forty-eight hours. In the first experiment the dogs were run for 47 days. The regeneration was good in both animals; if anything, better in the dog which received the extract (see Fig. 3). In the second experiment the full physiological dose was used, but the dog receiving parathormone died at the end of three weeks postoperatively from parathormone poisoning. In this animal the regeneration was distinctly poorer than in the concification was less than in the first series because of the shorter period of the experiment, but the poor result in the dog receiving extract was due either to the greater dose of parathormone, or more probably to the fact that a good approximation of the periosteum was not made because of the speed necessary to close a hole made in the parietal pleura during the rib resection.

In the third and fourth sets of adult dogs we observed very poor regeneration in all four animals. These were carried for four weeks post-

*This was very kindly supplied to us gratis by the Eli Lilly Co., of Indianapolis.

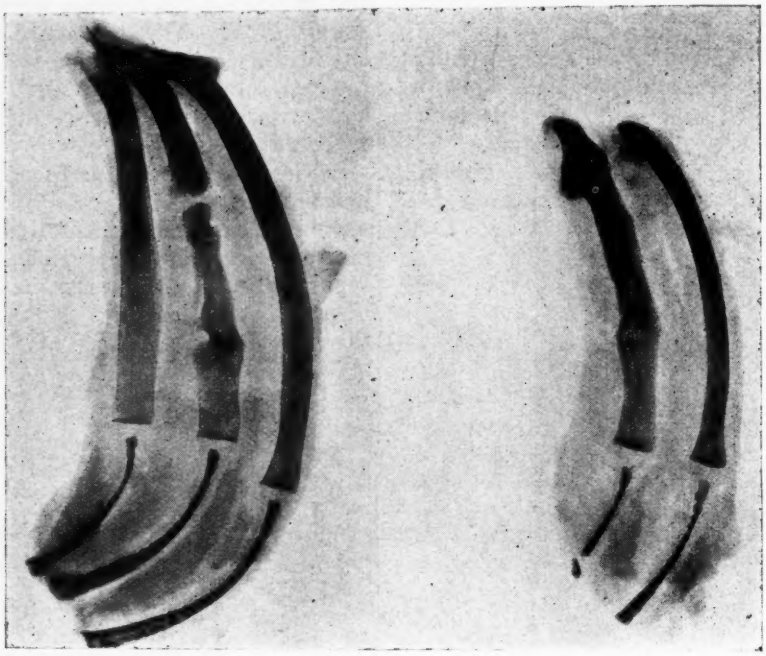


FIG. 3

Dogs 1 and 3—respectively—showing regeneration of rib resected subperiosteally 6 days after femoral lesion was produced. Specimens removed at autopsy 47 days after operation. Dog 3 (extract dog) shows somewhat better regeneration—but probably the difference is attributable to a more accurate reapproximation of the periosteum. Adjacent ribs included in photograph show no essential effect on normal bone as a result of parathormone administration.

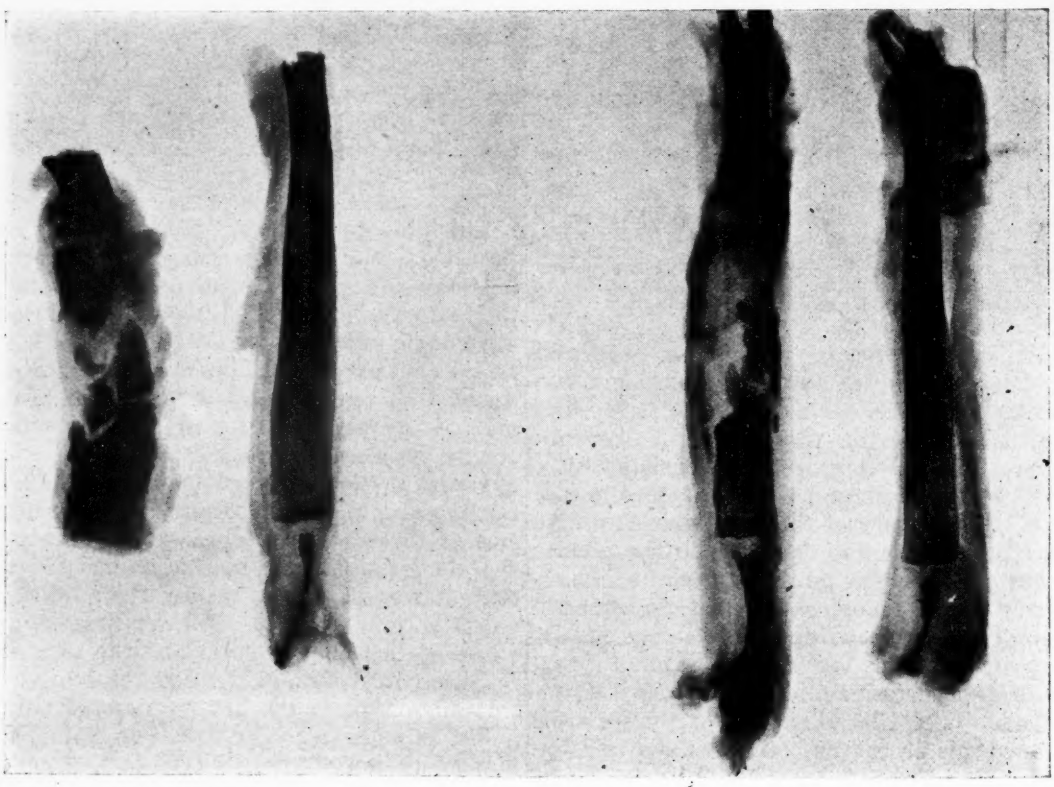
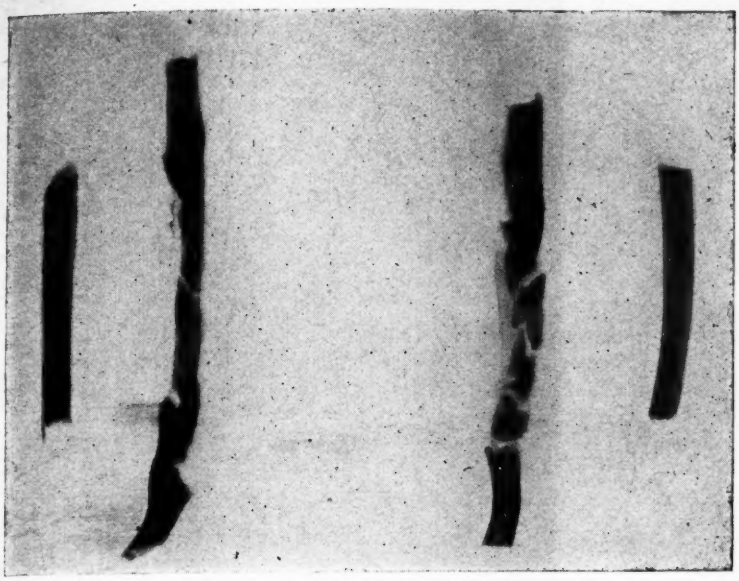


FIG. 4

Dogs 2 and 4 (autopsy specimens) respectively—treated same as Dogs 1 and 3 (see Fig. 3). Specimens removed at autopsy 16 days after operation. Regeneration therefore not so far advanced and distinctly poorer in the dog receiving parathormone. This was, however, probably directly attributable to a wholly inaccurate suture of the periosteum due to speed necessary to close an unintentional perforation of the parietal pleura. Here too the adjacent ribs show no difference in calcium deposition. The width of the ribs varies in the different figures because of the plane in which the ribs lay when the X-Rays were taken.



FIGS. 5 AND 5A

FIG. 5—Dogs 8 and 9 (pups—age 6 weeks) of the same litter, showing resected specimens, after regeneration, of subperiosteally resected ribs, with portions of adjacent ones for comparison, removed subperiosteally 27 days after operation. Dog 9 received 110 units of parathormone in 24 days post-operatively (½ full physiological dose). Dog 8 received no extract. The regeneration is not only obviously better in the control dog (the crack in the center was traumatically produced during resection), but during the removal of these specimens the periosteum was a well formed distinct membrane in Dog 8, and stripped easily, whereas in Dog 9 it was thicker, softer, and quite adherent externally to the regenerating rib.

FIG. 5A—Shows the regeneration in another rib of Dog 9 resected subperiosteally at the time of removal of the specimen in Fig. 5. This regeneration took place in 28 days, and during this second phase no extract was given—showing better regeneration than during a 27-day period with extract (see Fig. 5).

operatively, and the poor response is probably due to the fact that they were all old dogs.

A more clearly defined situation seems to exist in the case of young animals (see Figs. 4, 5, 5A, 6, 7 and 7A), for, in pups or adolescent dogs under the same conditions, there is a definite difference with respect to the facility with which calcium is deposited in newly forming bone. The extract inhibited calcification to a noticeable degree in four sets of experiments on dogs varying between three and ten weeks of age. In this series we had excellent controls with one exception (see Fig. 7), because in each case we had two animals from the same litter. In one experiment, furthermore, we obtained another type of control by using the same dog for a period of regeneration under the influence of the extract, followed by an equivalent period without it. Here, too, the regeneration was distinctly better during the second period when no extract was used.

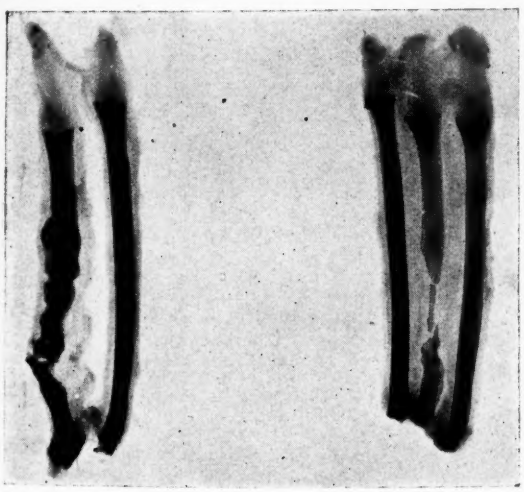


FIG. 6

Dogs 10 and 11 respectively (autopsy specimens), about one month old, each weighing 2.3 kilos, and from the same litter. Dog 10 was the control—and died 25 days after operation (autopsy disclosing no obvious cause of death). Dog 11 sacrificed 28 days after operation—having received only ¼ of full physiologic dose of parathormone in 22 days. Regeneration distinctly better in the control dog (the wrinkled appearance due to a too tightly pulled continuous periosteal stitch). Periosteal suture was carefully done in both animals. The apparent decalcification of the operated rib is about equal in both animals.

Although the number of experiments is small, it is reasonable to conclude that parathormone seems to delay the speed of deposition of calcium in regenerating bone of young dogs, whereas no clear evidence as to its potency in this or the reverse direction is available for adult dogs.

The practical significance of these observations is that the clinical use of the extract for delayed bone union is not based on any sound principle, can do little, if any good, and, in fact, may do harm. This is not in accord with Morel's findings with the use of his own extract, but there is no evidence that the extract he used in 1912 was potent or specific. It is further not clear why the removal of the gland should decalcify bone, as Erdheim claims, when we have this observation of the inhibition of calcification by providing what is probably the equivalent of an oversupply of the gland. But,

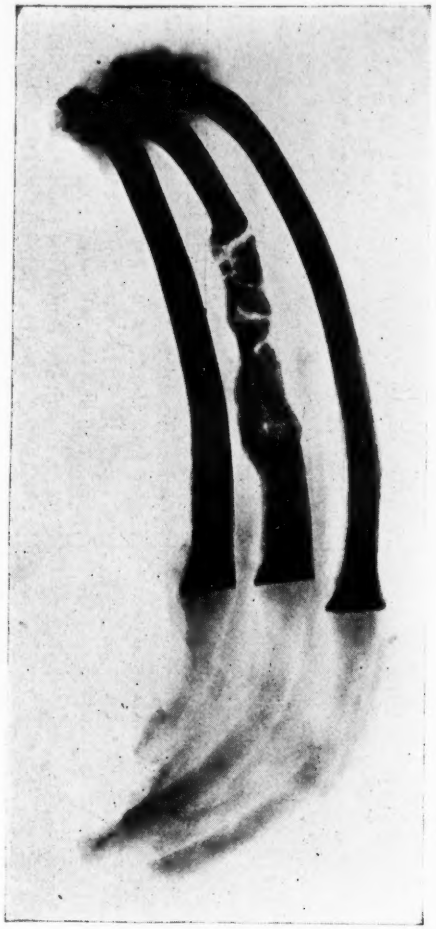


FIG. 7

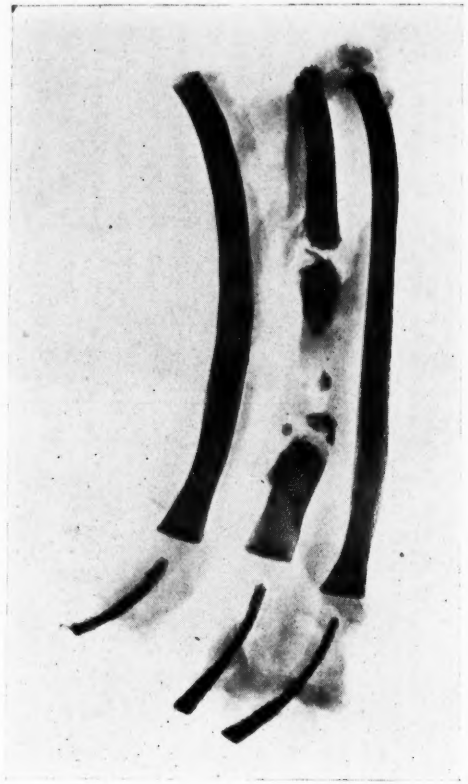


FIG. 7

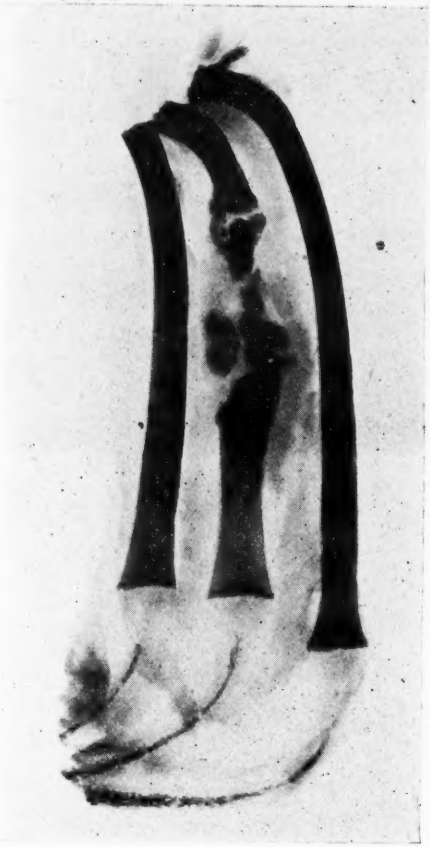


FIG. 7A

FIGS. 7 AND 7A

Dogs 17 and 18 respectively (autopsy specimens)—adolescent dogs. Dog 17 was the control. The conditions of this experiment were all in favor of the dog injected with parathormone because the control animal was sacrificed 17 days after operation owing to a prolapsed rectum and the parathormone dog was run for 14 days on a full physiologic dose and sacrificed 19 days after operation, though in good condition. In spite of these advantages, regeneration was distinctly poorer in the dog which received the extract.

In a similar experiment run at the same time the control animal acquired distemper immediately after operation and died 17 days later in a terribly dehydrated condition, having taken almost no food during the post-operative period. The regeneration was almost nil. If Dog 17, 1.7 kilos heavier than the extract dog of this experiment, is substituted as a control, the regeneration is again better, even though the extract dog's specimen was of 21 days duration as against 17 days for Dog 17. (Fig. 7A.)

In both experiments there is distinct decalcification, according to the X-Ray of the femur, radius, and ulna in the parathormone dogs by comparison with the control.

as already stated, this claim has not been confirmed. Whether these experiments support the assumption suggested by the case reports on osteitis fibrosa and osteomalacia, that the hypertrophied parathyroids found in these cases means decalcification from an oversupply of the glandular secretion, is purely a matter of opinion.

BIBLIOGRAPHY

1 Erdheim: Sitzungsb. Akad. Wissensch. in Wien., 1907, 116, 3, 311.
2 See Biedl, Innere Sekretion., 2nd Ed., Vol. 1, 109.
3 Erdheim: Frank. Zeitschr. f. Path., 1911, 7, 175, 238, 295.
4 Jovane and Vaglio: See Biedl, Innere Sekretion., 2nd Ed., Vol. 1, 109.
5 Korenchewsky: J. Path. and Bact., 1922, 25, 366.
6 Leopold and Reuss: Wien. klin. Wochnschr., 1908, 21, 1243.
7 Erdheim: Rachitis und Epithelkorpchen. Wien, 1914.
8 Ritter: Frank. Zeitschr. f. Path., 1920, 24, 137.
9 Pappenheimer and Minor: J. Med. Res., 1921, 42, 397.
10 Doyle: Science, 1925, 41, 118.
11 Tomaszewski: Frank. Zeitschr. f. Path., 1918, 21, 38.
12 Todyo: Frank. Zeitschr. f. Path., 1912, 10, 219.
13 Ritter: Frank. Zeitschr. f. Path., 1920, 24, 137.
14 Meyer: Frank. Zeitschr. f. Path., 1917, 20, 115.
15 Schmorl: München med. Wochschr., 1907, 54, 494.
16 Bauer: Frank. Zeitschr. f. Path., 1911, 7, 237.
17 Hohlbaum: Ziegler's Beitrage, 1912, 53, 91.
18 Goedel: Wien. klin. Wochnschr., 1925, 38, 246.
19 Mandl: Wien. klin. Wochnschr., 1925, 38, 1343.
20 Glison: Arch. ital. di Chir., 1924, 10, 238.
21 Hammett: Am. J. Anat., 1925, 35, 421.
22 Ogawa: Arch. f. exper. Path. u. Pharm., 1925, 109, 83, 300.
23 Dieterich: Arch. f. Klin. Chir., 1925, 136, 388.
24 Morel: Compt. rendu. Soc. de Biol., 1909, 67, 780.
Compt. rendu. Soc. de Biol., 1910, 68, 165.
Compt. rendu. Soc. de Biol., 1911, 70, 749, 877.
25 Gates and Grant: J. Exper. Med., 1927, 45, 115, 150.
26 Swingle and Nicholas: Am. J. Anat., 1925, 34, 469.
27 Collip: J. Biol. Chem., 1925, 63, 395.

PSYCHOPATHOLOGY AND TREATMENT OF THE PSYCHONEUROSES*

BY WILLIAM B. TERHUNE, M.D.†

I WISH to discuss with you an illness that causes unusual suffering, for a psychoneurosis not only makes the patient and his family suffer—it often distresses the doctor as well. I am especially appreciative of your invitation to address you today since it has helped me to attempt to define a psychoneurosis. It is difficult to find a satisfying definition of the condition and I have tried to evolve a criterion which might help to differentiate the psychoneuroses from other closely allied conditions.

First, it should be stated that at present I believe in the psychogenic causation of, and approach to the treatment of, the psychoneuroses. I make careful physical examinations, X-ray teeth, give gland extracts,—all in the hope that I may find a causative, or at least a contributing, physical factor. But at the present stage of our knowledge, the psychogenic explanations seem most logical and to produce the best results.

A psychoneurosis is multiform in its manifestation and causation. It is a functional disorder, not only because there are no physical causes explaining it, but more particularly because it is so intimately associated with the affective life of the individual. It is the result of a series of reactions, constitutional and environmental in nature. In studying an individual case it is necessary to determine what type of person the individual is constitutionally or inherently, then what conditioned responses have been built up as the result of environment and training, and finally, to search for precipitating situational factors. A large number of psychoneurotics have serious constitutional handicaps; some of these must be adjusted on a very low level of attainment. In the majority of cases the handicap is a result of individual experience such as emotional trauma, lack of training or poor training. Such patients are promising subjects for psychiatric reëducation. In every case there are precipitating situational factors which will explain the more severe symptoms, the nature of the symptoms being determined by such precipitating causes.

Many patients have mild mental disorders which are more closely related to psychotic than psychoneurotic conditions. Among these cases the mild affective reactions—such as those individuals showing cyclic mood alternations and the involutional depressions—are most common. Many of the so-called borderline cases might be classified as constitutional psychopathic states, while some others have schizophrenic tendencies with a paranoid trend. There is a large number of these mild mental patients of the three types just mentioned, who are able to adjust sufficiently satisfactorily to enable them to remain in the community. In a consideration of the psychoneuroses, these conditions are eliminated.

A psychoneurosis, then, might be described as a mild mental disorder with a tendency to chronicity. Hallucinations and delusions are absent, the emotional responses of the patient are relevant and the patient is completely in touch with reality—in fact he usually seems too acutely aware of reality and how it does or might affect him. The condition is usually roughly recognized because of the characteristic symptoms of neurasthenia, hysteria, the anxiety neuroses and psychasthenia.

The diagnosis of a psychoneurosis must not be made merely in the absence of physical findings, for such a condition may even co-exist with organic disease. The symptoms presented by the patient make us suspicious of a psychoneurosis, usually the chronicity strengthens such a suspicion, a careful study of the life history of the individual gives us some hint as to his preferred psychic reaction patterns—which are often psychoneurotic in nature—and a careful physical examination reveals the degree and quality of his physical adequacy. The most important diagnostic factor is a recent and relevant emo-

*Presented before the Franklin County Medical Society January 10, 1928, from the Austen Riggs Foundation.

†For record and address of author see "This Week's Issue," page 965.

tional strain. Such a factor is present in every case and is intimately tied up with the illness.

SOME USUAL PSYCHOPATHOLOGIC MANIFESTATIONS

The classification of the psychoneuroses as hysteria, neurasthenia, psychasthenia and anxiety neurosis (a traditional classification based on a certain grouping of symptoms) conveys very little idea as to the nature of the patient's illness. The logical approach to a better understanding is through a comprehension of some of the basic reactions underlying nervous illness.

The following reaction types are frequently encountered and seem to present the psychic basis of the patient's illness. Several of these reactions may be observed in an individual patient.

1. The escape and defense reactions.

1. The most frequent cause of a psychoneurosis is fear, and it is usually evidenced as an escape or defense reaction. For centuries human beings have built up methods of escaping difficulties and responsibilities. It would seem that certain of these reactions have become so deeply ingrained in the human race that they occur automatically, the automatism of escape usually being evidenced as an illness. Illness is a logical method of escape for two reasons: society grants a lower level of responsibility to people who are ill than for any other reason and, secondly, when people are in the throes of fear, the autonomic nervous system is so affected as to give rise to temporary symptoms of sickness. The escape reaction is usually an acute phase while the defense reaction is similarly motivated, usually occurring secondary to the acute phase, and consists of automatically building up self-protective reactions, often evidenced as chronic illness in order that fear may be avoided in the future.

2. Hypersensitization to sensation, suggestion, emotion.

2. Practically all who suffer from psychoneurosis are sensitive. A group of these patients are seen whose illness is characterized by a hypersensitization to specific sensations. In these cases the attention is fixed on the body and the patients are connoisseurs in sensation. Most of these hypochondriacal tendencies are based on fear, coupled with conditioned responses as a result of unpleasant sensory experiences. Suggestions may play an important rôle in causing such conditions .

3. The conditioned response.

3. The conditioned emotional or sensory responses are both factors in causing certain types of psychoneuroses. Pawlow demonstrated what he called the "conditioned reflex", showing how conditions merely present at the time of a reaction might in turn become a stimulus to set off this reaction. Certain psychoneurotic reactions can be explained in this way.

4. Anomalies of judgment.

4. Next to fear and sensitiveness poor judgment is the most frequent cause of a psychoneurosis. One of the outstanding facts about a psychoneurotic is his lack of judgment, which may be either the cause or a result of the illness. In either case it materially helps to continue the illness. A careful study of the life histories of patients reveals that there are some who have constantly shown poor judgment in meeting both the major and minor situations of life. As a result they are constantly inadvertently creating situations which upset them emotionally and make them ill. People can be taught to use good judgment. The psychiatrist treating the mild mental illnesses must often not only temporarily supply the better judgment for his patient but also must teach him how to use better judgment.

5. Repressions and inhibitions.

5. The intolerant repression of an emotion often results in a growth of intolerance towards the person or thing causing the repression, which will sooner or later result in an explosion. A repressed emotion is often manifested in a form quite different from its original state. Adult sex jealousy is often based primarily on the jealous individual's repressed desire to do exactly what he feels the other person is guilty of.

6. Affective-reality maladjustments.

6. There is another type of reaction frequently met with which might be termed the "affective-reality maladjustments". In these conditions we find an individual unconsciously straining to satisfy affective cravings which, under the circumstances of the realities of human existence, cannot be attained.

A good example of this is the ruminative phobics, who are constantly trying to analyze all factors underlying their phobias while they continue with their phobic demand for adult protection. Such conditions might be explained as follows: As a child we accept without question the ability of our parents to fully protect us,—in fact, they tell us, "Father will not let anything hurt or bother you." Thus the child is given the only complete protection he will ever experience—and it is only a sense of protection. As we grow up we constantly try to find such protection as we thought we had in childhood, and although the illusion of protection lapses into unconsciousness, the demand for it continues. As this desire for safety comes in touch with the dangerous realities of existence, an emotional perturbation may occur, leading to much thinking and an obsessive preoccupation which is the result of an unconscious effort to reconcile the affect of childhood with adult realities. There are many similar and less complicated affective-reality maladjustments—

such as those typified by reconciling childish ambitions with adult ability and opportunities.

7. Phantasy-hysteric reaction.

7. Books have been written on the hysteria reaction. One fact, however, is evident: people who have true hysteria are constitutionally different from those who do not. There is an hysterical type of personality. People who have hysteria have shown a life-long tendency to disassociate; they are day-dreamers and live much of their lives in phantasy. An hysterical reaction is an automatism of escape occurring in a person either constitutionally given to disassociation or trained to dissociate because of much day-dreaming.

8. Fright and phobias. Affect displacement.

8. The phobic reaction is a most difficult one to understand, for the basis of the fear is not that object or situation which the patient says and thinks he is afraid of. The phobics are constitutionally apprehensive, cyclothymic individuals. The phobia may be a psychic equivalent for a depression, apparently occurring without adequate reason, just as a depression at times seems to be a constitutional reaction. Again the phobia exists on the basis of a displacement reaction. A person may have a definite fear which his conscience is unwilling to admit to consciousness; as a result, the affect is displaced from its original object, and is expressed as an unreasonable and overpowering fear.

9. Reaction to maturity and dreaded involution.

9. Human beings have to take many hurdles in Life's obstacle race: puberty, college, financial responsibility, marriage, childbirth and old age are periods when psychoneurotic reactions are most apt to occur, and of these the middle-age reaction is the most serious. When women pass the menopause, they feel that their life is spent and when men are around fifty they have a similar reaction. Most human beings have wished to accomplish many things before they die and most of us in youth do not realize that it is not possible to accomplish most of our infantile desires. Therefore, when we reach middle age we have a sense of futility and disappointment, the source of much of which is unconscious, resulting often in a definite depression.

10. Sex motivated reactions.

10. Interference with the normal sex drive causes psychoneurotic reactions and this may be the result of a number of situations or conditions. There are a number of married women who suffer sexual excitement without experiencing the relief of an orgasm, who exhibit a rather characteristic excitable and overmobilized personality. Unfortunate and undigested sex experiences, as well as marital sexual maladjustments, may appear either as the cause or a symptom of a psychoneurosis.

11. Sickness as a method of warfare.

11. Husbands and wives, children and parents, and closely associated relatives often wage chronic warfare; should one of the combatants become ill during the course of the belligerency, the ready and all-subduing weapon of sickness is placed in his hands. Illness as a method of warfare is usually resorted to by one who is in danger of losing the battle.

12. Faulty habits of living.

12. Faulty habits of living, such as an improper daily balance of work, play, exercise and rest, lead to neurotic symptoms. Lack of relaxation and recreation is most apt to give rise to such transient conditions.

13. Acute grief.

13. There is a repressed acute grief reaction which is evidenced as a psychoneurotic illness. Persons suffering the loss of a loved relative may often become ill as a result of intolerance, self-pity or pent-up emotion. People who do not cry when someone they love dies are inhibiting a strong emotion and once again the inhibition may extend to all the mental content, causing a depression. Another manifestation of repressed grief is uncontrollable crying, the patient not experiencing a feeling of grief and not understanding the paroxysmal crying.

14. The reaction to a feeling of insecurity.

14. The feeling of insecurity may be the cause of a psychoneurosis. Many people have, for one reason or another, contracted a feeling of their inadequacy or inferiority. Under certain conditions of strain this gives rise to a chronic feeling of insecurity. This may create continual emotional perturbation, activating various psychoneurotic symptoms.

15. The hyper-reactive.

15. There are certain people who over-react to most situations. They are usually overmobilized, impatient and are unduly affected by stimuli. Their sense of proportion being disturbed, they dramatize and exaggerate all their reactions. These patients usually have a cyclothymic type of personality.

16. The hypo-reactive.

16. There are a few hypo-reactive individuals who seem very much out of touch with their environment. These are rather apathetic, uninterested, unaffected by outside stimuli; such patients have a schizophrenic type of personality.

The description of psychopathologic manifestations could be continued almost indefinitely, for there are an infinite number of possibilities. Seventy-five per cent. of the psychoneurotic illnesses, however, seem to be motivated by one or more of the reactions described. Other mech-

anisms might be formulated as explanation of the reaction types described and disciples of various psychological schools might be unwilling to accept some of these postulations. They are, however, an aid to me in understanding and treating patients and present what seems to be a logical working hypothesis.

TREATMENT

Many methods of treating the psychoneuroses have been reported; all are more or less efficacious. In recent years there have been no new therapeutic discoveries of consequence, but the psychoanalytic method of study and therapy has stimulated physicians to give time and thought to the effort of understanding their patients. This sympathetic understanding and interest existing mutually between physician and patient is the basis on which all good psychiatric work is accomplished. To this should be added the process of bringing a patient to an understanding of his condition and the causes underlying it, followed by a synthesis of these constructive factors in his life leading to a more satisfactory adaptation. The principal objects of modern psychotherapy will then have been attained.

There are various techniques tending to accomplish these aims, but since one purpose of this paper is to describe how the general practitioner may care for his patients in their homes, a simple technique will be described briefly.

First, evaluate the elements of organic illness and treat them. It is wise to get a patient organically well first, for it is difficult to treat an organic illness and functional difficulty at the same time. Get a complete history, not only of the patient's illness, but also his developmental history.

Then start with the patient on the basis of a functional illness and explain that nervousness is a real condition needing definite treatment. Be prepared to take time with these patients; count on giving them an hour a day for a month of intensive treatment.

Explain the object of treatment, i. e., to restore them to complete usefulness, and that to accomplish this their full coöperation will be needed. Put them on a written schedule, filling every minute of the day. Stress that you are trying to teach them to live, therefore budget their time in terms of work, rest, exercise and play every day; likewise encourage them to build up their physical health and strength. It is a good plan to give patients who are being treated at home a schedule in chart form on which they can keep track of how well they follow their schedule.

Encourage them to talk to you, listen to their stories, give them the relief of a sympathetic listener. Do not advise at first about their troubles; listen, take it under advisement, and they may later find the answer themselves. Most doctors are too busy and too accustomed to giving orders to listen to the troubles of a psychoneurotic. It is hard not to get impatient with these people—instead, try to understand them; as they talk, you can be trying to figure out why they are as they are.

Be careful not to give them fanciful explanations of the nature of their illness, such as autointoxication, glandular dysfunction or general debility, when the trouble is psychic, stick to your guns and see it through on that basis.

Much nervousness is poor mental hygiene. Give your patients a course in mental hygiene. Show them that life is a matter of adaptation. Give them some comprehension of how the central nervous system helps them in this; explain to them how consciousness affects conduct. Give them some understanding of the nature of emotions, instincts and affect.

Explain to them the nature of functional illness and the formation of such symptoms. These symptoms must be treated as realities and patients in general need to be shown why functional symptoms have not the same significance as those caused by appreciable organic lesions. Show them how fatigue may be a matter of mood and emotion and that many physical symptoms are no more than reverberations of the autonomic nervous system.

Teach them to discount sensitiveness, emotions and somatic symptoms; to do this by understanding such symptoms and, therefore, to live objectively, not subjectively.

Convince them that it is unwise to fight their functional symptoms, help them to be more tolerant to such symptoms, then accept them and be willing to live with them. Explain that by so doing their attention will no longer dwell on themselves and their symptoms and, therefore, the symptoms will cease to exist.

With this background, take up their individual emotional experiences, and help revalue them. Investigate their personal difficulties and help them to solve them. And in general assist them to work out an adjustment on a level for which they are suited. Help them to find new interests.

Sensible human beings all have a feeling of inferiority. This is often intensified by illness, especially nervous difficulties. Therefore, always be kind, bolster up their self-respect. To scold a psychoneurotic is both brutal and poor technique.

Occupation is an important part of therapy in treating psychoneurotics. Keep them busy, show them that it is not sufficient to merely accomplish things but that they must learn the joy and satisfaction of skilful craftsmanship. A person who has a psychoneurosis needs some occupation, as a temporary therapeutic measure, which is stimulating to his intelligence and which will arouse new interests; for this reason it is often wise to have such patients take up some new form of work which may either supplement their regular jobs or temporarily take

the place of them. Weaving, carpentry, gardening, art, music, correspondence courses, learning Braille and scientific research all offer opportunities for occupational therapy.

The psychoneurotic, like most people, needs new interests; help him to find these and show him that he must constantly be on the alert for all new interests which will help him to grow, rather than fall into a mental rut which leads to preoccupation and fixation on self.

And if fear is the greatest cause of the psychoneurosis, teach your patients to live adventurously. Help them to understand that fear may produce such thrills as to put new interest in living.

The physician treating a psychoneurotic patient must not be discouraged at his slow progress. It would seem that a period of absolute failure precedes most recoveries. It has been said that just as the patient is turning the corner on the road to recovery, he is most apt to feel worse; when the patient feels ready to give up, that is the time that the doctor has the best chance to get him well.

MINIM METHOD FOR ANALYSIS OF GASTRIC CONTENT

BY MOSES EINHORN, M.D.*

THE method about to be described is based primarily upon a procedure which I recently devised for analyzing gastric content without using more than two or three drops of the specimen.† It is therefore necessary to give a brief summary of this last-named method in order to understand how it was derived from the old method and also to indicate wherein it differs from the one which I am now presenting. This is best done by describing how an analysis of gastric content would be made under each of the three methods referred to, namely, the old method, my "simple and rapid method" and the new "minim method".

OLD METHOD: Let us imagine that 3 c.c. of decinormal sodium hydroxide had to be added to 10 c.c. of the gastric specimen in order to change to yellow the reddish color caused by the previous addition of Toepfer's solution to the specimen. Multiplying by ten, we get the number of cubic centimeters of free acid in one hundred cubic centimeters of the particular specimen under examination to be thirty, or F equals thirty. Let us now suppose that two cubic centimeters of the decinormal solution of sodium hydroxide are required in order to bring about a scarlet color in this specimen after the addition of a few drops of phenolphthalein. Again multiplying by ten, we find the number of cubic centimeters of other acids present in this specimen to be twenty and adding this reading to the previous one, we find that the total acid in one hundred cubic centimeters of the specimen under examination is fifty, or T equals fifty.

SIMPLE AND RAPID METHOD: Using the hypothetical test outlined above as a basis and bearing in mind the fact that the pipette used in this test will deliver twenty minims to the cubic centimeter, I will now proceed to indicate in what fashion I derived this "simple and rapid" method. This is done by means of the following equations, in which the minim measurements are substituted for the cubic centimeters and the decinormal sodium hydroxide is replaced by N/100 sodium hydroxide and that, in turn, by the N/200 and the N/400 sodium hydroxide. Thus:

3 c. c. of N/10 NAOH are required to neutralize 10 c. c. of gastric content giving the final reading for free acid in 100 c. c. of gastric content as F equals 30.

(1) or 60 minims of N/10 NAOH are required to neutralize 10 c. c. of gastric content giving F = 30.

(2) or 6 minims of N/10 NAOH are required to neutralize 1 c. c. of gastric content giving F = 30.

(3) or 6 minims of N/10 NAOH are required to neutralize 20 minims of gastric content giving F = 30.

(4) or 3 minims of N/10 NAOH are required to neutralize 10 minims of gastric content giving F = 30.

(5) or .3 minims of N/10 NAOH are required to neutralize 1 minim of gastric content giving F = 30.

(6) or 3 minims of N/100 NAOH are required to neutralize 1 minim of gastric content giving F = 30.

(7) or 1 minim of N/100 NAOH is required to neutralize 1 minim of gastric content giving F = 10.

Now, substituting N/200 NAOH for N/100 NAOH in equation (6) we get:

(8) 6 minims of N/200 NAOH are required to neutralize 1 minim of gastric content giving F = 30.

(9) or 1 minim of N/200 NAOH is required to neutralize 1 minim of gastric content giving F = 10.

Again, substituting N/400 NAOH for N/100 NAOH in equation (6) we get:

(10) 12 minims of N/400 NAOH are required to neutralize 1 minim of gastric content giving F = 30.

(11) or 1 minim of N/400 NAOH is required to neutralize 1 minim of gastric content giving F = 2½.

Using the results obtained in equations No. 7, No. 9 and No. 11, we came to the conclusion in this "simple and rapid method" that the following readings could be used as a standard:

*For record and address of author see "This Week's Issue," page 965.

†Einhorn, Moses: "Simple and Rapid Method for Quantitative Analysis of Gastric Content", (Medical Journal and Record, November 16, 1927).

Every time we used one minim of N/100 NAOH to neutralize one minim of gastric content, we could add to the final reading F equals 10 if the Toepfer reagent had been used, or T equals 10 if the phenolphthalein reagent had been used. Similarly, each drop of N/200 used would add five to the final reading and each drop of N/400 used would add two and one-half to the final reading. It will be noted that in the example given above pipettes which delivered 20 minims to the cubic centimeter were used. However, the standards which we have obtained are not dependent upon the use of that particular size of pipette. Pipettes which deliver any number of minims to the cubic centimeter may be used but what is required is that the same size and type of pipette should be used for the neutralizer as is used for the gastric content.

MINIM METHOD: In the old method and in the "simple and rapid method", it was necessary to apply the reagent and the neutralizer separately. In addition to complicating the test, this procedure was disadvantageous in that too strong a concentration of the reagent would often be added to the specimen and thus mitigate the accuracy of the results. Another difficulty was that the technician, through lack of a suitable standard, could not readily tell when the mixture had attained the proper color. It occurred to me that if we could mix the reagent and the neutralizer in the proper proportions and still retain the standard readings used in the "simple and rapid method", the above-mentioned difficulties would be eliminated. After considerable experimentation, I have found the proper proportions for the various reagents and neutralizers and have incorporated them in six solutions, the names and compositions of which follow:

Yellow Solution No. 1—1 minim of Toepfer's Solution to 5 c. c. of N/100 NAOH.
Yellow Solution No. 2—2 minims of Toepfer's Solution to 5 c. c. of N/200 NAOH.
Yellow Solution No. 3—3 minims of Toepfer's Solution to 8 c. c. of N/400 NAOH.

Scarlet Solution No. 1—1 minim of Phenolphthalein to 12 c. c. of N/100 NAOH.
Scarlet Solution No. 2—1 minim of Phenolphthalein to 7 c. c. of N/200 NAOH.
Scarlet Solution No. 3—2 minims of Phenolphthalein to 9 c. c. of N/400 NAOH.

Since the creation of these solutions did not destroy the standard readings referred to above, we can now say that for every drop of No. 1 Solution (Yellow or Scarlet) used in this test the final reading is increased by ten, for every drop of No. 2 Solution used (Yellow or Scarlet) the final reading is increased by five and for every drop of No. 3 Solution used (Yellow or Scarlet) the final reading is increased by two and one-half.

Before outlining the procedure followed in this "minim method", it is again necessary to caution the technician to use the same type and size pipette for the gastric content as is employed for the neutralizer because the standard readings given above apply only when the same size minim is used throughout the test. In this connection, I have found it best to use pipettes which have their ends bent in a 60° angle, for, by holding these pipettes in a horizontal position, the technician can be sure that the pipette is being held in that position in which it will always deliver the same sized drop.

METHOD: (A) TO DETERMINE THE AMOUNT OF FREE ACID: Using the type of pipette referred to above, the technician takes one clear drop of the gastric specimen and places it in the special paper tray. (See Fig. 1.) When the Yellow Solution is first applied, a reddish color is obtained if there is free acid present. The next few applications will usually intensify this reddish color while succeeding applications of the Yellow Solution will gradually cause the specimen to take on a reddish-yellow color. At this point, the technician replaces the Yellow Solution No. 1 with the weaker solutions (No. 2 and No. 3) and uses these until the specimen is exactly the same color as the Yellow Solution. If the specimen turns the desired color immediately upon the application of the first drop of Yellow Solution No. 1 it is usually an indication that the specimen contains no free acid. However, the examiner should place another

FIGURE 1.

drop of the specimen in a new tray and proceed to test with the weaker solutions, No. 2 and No. 3, in order to detect and measure whatever small amounts of free acid may be present. The amount of free acid in the specimen is determined by noting how many minims of the various Yellow Solutions were required to give the specimen the same color as the Yellow Solution. For each drop of Yellow Solution No. 1 the final reading is increased by ten and in like fashion the technician adds five for each drop of Yellow Solution No. 2 and two and one-half for each drop of Yellow Solution No. 3. Thus, if the technician uses two minims of Yellow Solution No. 1, one minim of Yellow Solution No. 2 and one minim of Yellow Solution No. 3, the final reading will be "Free acid = 27½."

(B) TO DETERMINE THE TOTAL ACIDITY: The technician takes another clear drop of the gastric specimen and places it in a new paper tray. At first when the Scarlet Solution No. 1 is applied, its color will be quickly lost in that of the specimen. The technician continues adding Scarlet Solution No. 1 until the specimen begins to turn scarlet. At this point, he replaces the Scarlet Solution No. 1 with the weaker solutions, No. 2 and No. 3, and applies these until the specimen is almost the same color as the Scarlet Solution. As in the test for free acid, the same standard readings apply, i. e., ten for each minim of Scarlet Solution No. 1, five for each minim of Scarlet Solution No. 2 and two and one-half for each minim of Scarlet Solution No. 3. Unlike the procedure under the old method, this reading is not added to the previous reading for free acid but stands alone as the final estimate of the total acid in the specimen under examination. Thus, if in bringing about the change to scarlet the examiner used four minims of Scarlet Solution No. 1, three minims of Scarlet Solution No. 2 and one minim of Scarlet Solution No. 3, the final reading for total acid would be 57½ or T = 57½.

The standard readings which form the basis of this method will be found to hold true in all instances provided the technician observes the following simple precautions: Care should be taken to use the same type and size of pipette for applying the neutralizer as is used in applying the specimen. Also, the technician should use pipettes whose tips are bent at a 60° angle for by holding these in a horizontal position the technician will always be able to obtain the same sized drops. Both the Yellow and the Scarlet Solutions should be kept in brown bottles so as to prevent the light from affecting them. This will not prevent the technician from using these solutions as color standards for he will be able to observe the proper color in the transparent pipettes with which these solutions are applied. Both the specimen of the gastric content and the solutions should be kept at room temperature.

Summing up the advantages which are possessed by the Minim Method, I find that it is preferable to the other methods for the following reasons:

1. By combining the reagent and the neutralizer in one liquid, the necessity of separately applying the reagent is eliminated.
2. Because the Yellow Solution and the Scarlet Solution can be used as standards in determining whether the proper color has been obtained in the specimen under examination, the possibility of error in the readings due to faulty judgment of the color changes is reduced to a minimum.
3. The possibility of error due to too strong an application of the reagent to the specimen is eliminated by combining the reagent and the neutralizer in the proper proportions.
4. No filtration is now necessary as it is always possible to secure a number of clear drops of the specimen which do not contain any particles.
5. No matter how small a specimen is secured, it is now possible to make a complete qualitative and quantitative examination of the gastric content.
6. In a fractional examination, instead of removing five or ten c.c. at the various intervals, we need now only remove a few drops. In this fashion, we do not remove too much content from the stomach and thereby make our plotted curve a more accurate and reliable picture of the gastric digestion.
7. This method is more simple than the previous methods, less calculations are required and it does not involve any measurements. This fact is especially noticeable in the fractional examination when a series of tests is necessary.
8. Under the old method, even though the beaker used is carefully rinsed between successive tests, some part of the content or neutralizer often remains with the result that the readings are affected. These possibilities of errors are eliminated in my method as an individual paper tray is used for each test.

DIFFERENTIAL DIAGNOSIS IN PULMONARY DISEASES

(With Particular Reference to the Diagnosis of Pulmonary Tuberculosis)

BY MARK H. JORESS, M.D.*

THE purpose of this paper is to review the various pulmonary lesions, and some non-pulmonary diseases which resemble tuberculosis, and to cite several cases illustrating errors in diagnosis.

The diseases to be considered can be grouped as follows:—

Group I: Bronchitis, bronchiectasis, abscess,

*For record and address of author see "This Week's Issue," page 965.

bronchial asthma, bronchopneumonia and lobar pneumonia, pulmonary fibrosis, and pneumoconiosis.

Group II: Neoplasm, cysts, mycosis, spirochetosis, and syphilitic lung.

Group III: This group, outside the lung fields, includes heart disease, thyroid disease, and accessory nasal sinus disease.

Bronchitis and Bronchiectasis: Chronic bronchitis, with a gradual dilatation of the bronchi, leads to the advanced condition of bronchiectasis and the formation of bronchiectatic cavities. The most common factors in etiology are a preceding pneumonia, influenza, and whooping cough. The latter, in particular, is responsible for many cases of bronchiectasis in children. The symptoms common to both conditions are cough, shortness of breath, pain in the chest, and expectoration. Expectoration is periodically profuse in a case of bronchiectasis, and usually of a foul odor. Bloody sputum occurs in both conditions, especially in bronchiectasis, where the amount of blood lost may even prove fatal[1].

Quite unlike the tuberculosis case, the patient with bronchitis does not show symptoms of a toxemia, and there is no loss of flesh. Save for the cough, which is annoying at times, these patients have no symptoms and go about their work as though nothing was the matter with them. Even the patient with bronchiectasis maintains his body weight and is not toxic, as a rule, in spite of the presence of pus in the bronchial tree. When, however, drainage of pus is interfered with and absorption takes place, a toxemia supervenes as evidenced by fever and malaise.

As for physical signs, the lung parenchyma shows no infiltrative changes; no dullness or changes in the respiratory note. The abnormal signs present are sibilant and sonorous rhonchi mixed with coarse mucous râles heard over one or both lungs, and usually limited to the base.

In the exceptional case of bronchiectasis accompanied by ulceration into the lung parenchyma dullness, bronchial breathing, and coarse mucous râles are the signs met with. These signs are basilar. A characteristic sign in bronchiectasis is clubbing of the fingers.

The tubercle bacillus is not found on smear, culture or guinea pig study of the sputum. The prevailing organisms are the influenza bacillus, the pneumococcus, and the staphylococcus. Lipiodol injected into the bronchial tree is of invaluable assistance in the diagnosis of a suspected case of bronchiectasis.

To summarize: A history of repeated attacks of bronchitis accompanied by profuse expectoration, particularly of a foul odor, negative for the tubercle bacillus, with clubbing of the fingers, with no signs of parenchymatous lung infiltration in a patient in good physical condition, warrants a diagnosis of bronchitis or bronchiectasis rather than tuberculosis.

Lung Abscess: It is caused by aspiration of foreign bodies; is a complication of operative procedures; and is seen to follow in the wake of an unresolved pneumonia. Acute abscess is ushered in by pain in the chest, cough, fever, chills, sweats, and prostration. There is no expectoration until the abscess has ruptured into a bronchus, then foul sputum appears. The chronic abscess with adequate drainage may be devoid of symptoms other than cough and expectoration.

The cough in lung abscess is quite different from that in tuberculosis; it is explosive in character. Clubbing of the fingers is a prominent sign and appears quite early. Years may elapse before this sign is seen in tuberculosis, then usually in the far advanced case with cavity formation. The common seat of pathology in abscess is at the base, while in tuberculosis the upper portion of the lung is usually affected. Physical signs of abscess are dullness, bronchial breathing, and localized coarse râles.

The X-rays are of distinct aid in mapping out the exact location of the abscess. They likewise indicate the structure of the abscess, whether it is a single cavity or multilocular. The bacteria found commonly are pneumococci, streptococci, and staphylococci.

Summarizing, abscess is acute in onset; tuberculosis is insidious. The sputum is foul in the former and rarely so in the latter. Abscess usually elects a lower lobe while tuberculosis is most likely to involve an upper lobe or lobes.

Bronchial Asthma: It is confounded with tuberculosis because of weakness, dyspnea, cough, expectoration, and occasional bloody sputum. Differential diagnosis rests upon the elimination of factors of allergy or septic foci responsible for bronchospasm.

As for physical signs, there is no evidence of infiltration of the lung parenchyma. Dullness and voice changes are lacking. The signs commonly elicited are: emphysema, hyperresonance and sibilant and sonorous rhonchi ("wheezing") scattered throughout both lung fields. If asthma is superimposed upon a tuberculous process the latter may be readily overlooked. Signs of tuberculosis may be demonstrated only during a remission from bronchospasm.

Bronchopneumonia and *Lobar pneumonia:* The similarity between these diseases and tuberculosis is very marked, but the clinical history is quite different. The former are acute in onset in a previously well person, whereas phthisis is insidious. The pneumonias are ushered in by a chill, accompanied by fever, headache, pain in the chest, dyspnea, and prostration. On the other hand, tuberculosis shows almost invariably a prodromal period with the following common symptoms: loss of strength and sustained endurance, loss of flesh, afternoon rise in temperature, indigestion and fatigue. The leucocyte count is usually high in pneumonia but rarely so in tuberculosis.

The characteristic physical signs in a bronchopneumonia are subcrepitant râles heard at both bases often associated with sibilant rhonchi. Tuberculosis, on the other hand, is confined usually to the upper lung fields with signs of dullness, increase in voice conduction, and râles. The signs of a lobar pneumonia are dullness, bronchial breathing, and if resolution has set in, râles; and with rare exceptions this pathology is confined to the base. The signs just described are occasionally met with in tuberculosis. We have then a tuberculous pneumonia. However, the two may be separated readily when remembering that pneumonic phthisis has almost invariably an antecedent period of ill health which does not occur in a pneumonia.

Pulmonary Fibrosis: This condition is seldom thought of, although it bears a direct relationship to the last epidemic of influenza. The latter disease is responsible for a chronic lung fibrosis in the adult, and is observed to follow pneumonia and whooping cough in children. In a study of this condition Atkinson[2] has observed the following features: "Recurrent cough, worse in the winter months, always productive. Children seldom have foul sputum and never in large quantities. Temperature and pulse are normal and there is no tendency to lose weight."

As for physical sign, both sides of the chest move equally; areas of dullness over one or both bases with râles confined usually to the base are the most frequent signs elicited. The upper lung fields show no dullness, voice changes, or râles, which are the common signs in tuberculous disease.

Summarizing, these patients do not show the progression in their disease which we see in tuberculosis. As a rule, they appear in good health. The X-rays show exaggerated lung markings but these do not involve the apices or the lung periphery. Above all, these patients date the onset of the present illness to a preceding influenza, pneumonia, or whooping cough.

Pneumoconiosis: This disease is observed in stone cutters, fertilizer makers, and grain handlers. Silicosis, the most common of the entire group of dust diseases, is an insidious fibrotic lung disease characterized by dyspnea, cough, and expectoration. The early silicotic lung changes are mild and are not recognized because there are slight or no symptoms present. As the disease progresses, with more and more of the lung alveoli impregnated with silicon particles, dyspnea sets in with a tendency to grow steadily worse. In time the lungs are invaded by pathogenic organisms and an infectious bronchitis is set up accompanied by malaise, loss of strength, and a variable degree of toxemia. The tubercle bacillus may become the invader and soon we have developed an "infected silicosis", as silicosis with phthisis superimposed upon it is called.

The physical signs of silicosis are variable[3]; there are areas of dullness, most commonly at the bases, with absence of breath sounds, as well as areas of bronchial breathing. Râles may or may not be heard. When present they are confined to the bases rather than the upper lung fields as seen in phthisis. As the disease progresses emphysema is rather constant. The latter physical finding is rarely seen in tuberculosis. Rather characteristic of silicosis is the nearly constant bilaterality of lung involvement.

When complicated by tuberculosis the diagnosis of silicosis is more difficult, especially in the absence of a bacillary sputum. The X-rays may afford the most reliable data in establishing a diagnosis.

Summarizing, silicosis carries with it an occupational background of silicon dust. Physical signs in silicosis are usually basilar; tuberculosis elects, as a rule, an upper lobe. Areas of dullness with absence of breath sounds as seen in silicosis are not met with very commonly in tuberculosis infiltration. The presence of râles over the bases is the unusual finding in tuberculosis. Emphysema is a common sign in advanced silicosis, and is rarely met with in tuberculosis. In a general way, in any patient with respiratory symptoms and an occupational background, that speaks for the inhalation of silicon laden dust, silicosis should be kept in mind.

Neoplasm of Lungs: A neoplasm in this locality simulates tuberculosis very closely. The common symptoms observed by one group of writers[4] are: Chest pain, worse at night; cough, expectoration, dyspnea, hemoptysis, hoarseness, weakness, and loss of weight. The physical signs found by the writers mentioned are: respiratory lagging of the affected side of the chest; impairment of the percussion note or complete dullness. Breath sounds are diminished or completely absent. This absence of breath sounds is quite contrary to what we find in the average case of tuberculous infiltration. The signs elicited in neoplasm are confined most commonly to the region of the hilus or the base of the lung. The X-rays are of the utmost importance in the study of these cases[5] since the physical signs are inconstant and may occur in any part of the lung.

Lung Cysts: The echinococcus cysts are the most common. The usual signs are diminished or absent breath sounds with dullness. The extent of the signs varies with the size of the cysts. They usually go unrecognized until they rupture into a bronchus, when cough, purulent expectoration, and hemoptysis appear.

In contradistinction to the signs of diminished or absent breath sounds with dullness, tuberculosis almost invariably shows an increase in breath sounds when dullness is present. Then, again, the classical râles heard in the upper lung fields in tuberculous disease are not met with in cysts of the lung.

Eosinophilia is an important differential guide, whereas the isolation of the parasites' hooklets establishes the diagnosis.

Pneumomycoses and Bronchopulmonary Spirochetoses: These parasitic diseases of the lungs resemble tuberculosis so closely that the latter is the diagnosis commonly made. Castellani[7] says of these infections: "In mild cases there are types of slight bronchitis with mucopurulent expectoration in which the fungi are found. In severe cases the patient presents symptoms suggesting phthisis with hectic fever and hemorrhagic expectoration." Diagnosis depends upon the isolation of the fungus or spirochete in question.

Syphilitic Lung: The physical signs may be those of a pulmonary fibrosis or an ulcerative tuberculosis. In the former type the common symptoms are cough, mucopurulent expectoration, and dyspnea. In the latter variety, cough, purulent expectoration and hemoptysis occur. Differential diagnostic criteria are: for syphilis, a positive Wassermann reaction in the absence of a bacillary sputum; and for tuberculosis, a negative history of primary or secondary specific lesions, negative blood, and positive sputum.

THE NON-PULMONARY GROUP OF DISEASES: 1. *Heart Disease.* Under this heading are considered myocarditis with failing compensation and mitral stenosis. The following symptoms are common to both: cough, expectoration, dyspnea, digestive symptoms, and hemoptysis. The last symptom occurs more frequently in mitral stenosis. Symptomatically we are dealing with diseases which resemble tuberculosis very closely. However, there are no physical signs to be found consistent with parenchymatous lung changes. Râles, when present, are confined to the base. The X-rays throw much light on the pathology at hand: the lung parenchyma is clear, while the so-called "lung markings" are increased due to stasis in the pulmonary circulation. Moreover, a careful physical examination will reveal evidence of cardiac disease.

2. *Thyroid Disease:* The toxic adenoma with symptoms of tachycardia, fatigue, nervous irritability, loss of weight, sweating, and fever simulates tuberculosis very closely. Though at times a patient with early phthisis may show an increased basal metabolism, the latter finding is a helpful guide to the diagnosis of thyroid disease. Moreover, the usual clinical manifestations of Graves' disease will point to the correct diagnosis. The lung parenchyma in thyroid disease shows no changes on physical examination or with the X-rays.

3. *Sinusitis:* Chronic disease of the accessory nasal sinuses is frequently accompanied by lung infection resulting in bronchitis and even bronchiectasis[8], with symptoms of cough, mucopurulent expectoration, hemoptysis, and fever. The physical signs are confined to the base, though, rarely, apical changes take place suggesting a tuberculous process. Differential diagnosis rests upon negative signs for tuberculosis, absence of tubercle bacilli on smear, culture, and guinea pig inoculation[9], and physical signs confined to the base with demonstrable paranasal sinus disease by the X-rays.

Having reviewed the various diseases resembling tuberculosis, let us consider a diagnosis of the latter. In this connection a carefully taken history is of the utmost importance. A history of contact with an open case suggests not only infection but potential disease as well. Though the manifestations of phthisis are variable, a history of loss of strength, lack of sustained endurance, a morning cough, indigestion, nervousness, and slight elevations in temperature are very suggestive. The symptoms just mentioned are not particularly striking, and are easily overlooked until some such episode as a hemoptysis takes place. Hemoptysis is one of the cardinal signs of phthisis.

Attacks of pleurisy unassociated with the acute diseases like influenza or pneumonia should be considered tuberculous in nature, and the patient should be observed for other evidence of phthisis. If an effusion accompanies an attack of pleurisy we have another cardinal sign of tuberculosis.

As for other symptoms: Though a loss of weight occurs in many other conditions, a steady loss of flesh deserves careful consideration. Night sweats are alarming to the patient. Hoarseness, because it is insidious as a rule, causes no alarm and is often overlooked unless it is part of a far advanced pulmonary lesion. This symptom may be due to a tracheo-bronchial gland enlargement with laryngeal nerve pressure, or is the result of a tuberculous laryngitis.

Physical signs in phthisis vary from such slight phenomena as suppressed breathing, slight increase in whisper and spoken voice, and harsh and prolonged expiration, to such marked signs of pulmonary infiltration as bronchial breathing with râles. Râles found above the third rib and fifth dorsal spine which do not clear up with cough constitute another cardinal sign of phthisis. Though the upper portion of the lung is the usual initial seat of tuberculous pathology, basal tuberculosis is not rare[10].

A positive X-ray and positive sputum are the last two of the five cardinal signs of phthisis. The X-rays are of paramount importance in the study of border line cases, that is, those cases which have a suspicious symptom complex but no definite physical signs. They serve here as an aid in establishing a positive diagnosis. The isolation of the tubercle bacillus is proof positive of the disease, yet it is of value only in a positive way since many cases of manifest disease never show a bacillary sputum in the routine smear examinations.

COMMENT

No doubt many cases are diagnosed as tuberculosis on insufficient evidence. It is equally true that many obvious cases are overlooked. It is at times very difficult to draw the line between the suspicious and positive cases, especially in the absence of the cardinal diagnostic signs.

If differential diagnosis is entered into systematically, a diagnosis of tuberculosis can be made in the great majority of cases with a surprisingly small margin of error.

REVIEW OF CASES

CASE I. Male, age 46, baker. *F. H.* One sister died of tuberculosis. *P. H.* Chronic "catarrh". *P. I.* In good health up to five weeks ago, when he caught cold, which was followed by fever, chills, pain in left chest, headache, cough, and expectoration. Fever left him at the end of a week, and he was convalescing. Admitted to the Rutland State Sanatorium with the following symptoms: cough, expectoration, and pain in left chest on deep breathing. *P. E.* Pleural rub left base in back. Otherwise essentially negative. *Laboratory reports:* Sputum negative on sixteen consecutive examinations. Smears of sputum showed: gram positive cocci, large gram positive diplococci. By culture: staphylococci and streptococci. *X-ray report:* "Seventh interspace posteriorly on the left shows an irregular density. Apices and lung fields clear. *Impression:* Unresolved pneumonia". Subsequent films showed a clearing up of the process. Discussion: The man was in good health until he developed the acute respiratory illness. Fever subsided as suddenly as it came. The physical findings on entrance were those of a pleuritis. The negative apices and lung fields suggested a non-tuberculous lesion. The diagnosis of tuberculosis was made on insufficient evidence. *Discharge Diagnosis:* "Unresolved Pneumonia".

CASE II. Male, age 37, laborer, chronic alcoholic. *F. H.* Negative. *P. H.* Unknown illness at 19 years, lasting three and one-half months accompanied by cough. Shortness of breath for many years. *P. I.* Steady, hacking cough at times productive, during past two years. Has had a scanty amount of sputum, loss of strength, and increasing dyspnea on exertion. On two occasions, two months before admission, raised a mouthful of blood. In the meantime, expectoration was increasing, was more short of breath, and cough was worse, especially at night. Has had night sweats and chills. Has lost eighteen pounds in weight. *P. E.* on entrance to the Rutland State Sanatorium: Orthopnea and moderate cyanosis. Well developed and well nourished. Chest: heart enlarged, action regular but rapid, sounds weak and distant, and a systolic murmur present at the apex transmitted into axilla. Pulse of poor tension and volume. Lungs: percussion note impaired over bases; no changes in breath sounds or voice conduction; numerous mucous râles over bases with and without cough. *Laboratory Reports:* No tubercle bacilli found on twenty-four examinations of sputum. *X-ray report:* "Heart shadow increased in its transverse diameter; bronchial tree prominent; bases hazy; upper lobes each lung clear. *Impression:* Heart Disease." *Progress notes:* Cough, expectoration, and dyspnea subsiding under digitalis therapy. *Discussion:* The alcoholic history in the case may be a factor in damaging the man's myocardium. He developed cough, expectoration, dyspnea, and hemoptysis. Had sweats and chills. There was no evidence of lung infiltration. Thus far the myocardium had been overlooked. The murmur at the apex was due to a mitral insufficiency induced by cardiac dilatation. The râles were due to passive congestion. The negative sputa, basal findings, X-rays negative for tuberculosis, and the presence of abnormal heart signs were against tuberculosis. *Discharge Diagnosis:* "Myocarditis with broken compensation."

CASE III. Female, age 38, housewife, one child, no miscarriages. *P. H.* Measles, chicken pox, appendectomy and hysterectomy. Influenza 1919. *P. I.* Dates back to the influenza, which left her with a persistent cough, at times followed by the raising of large quantities of sputum, not foul. Has been a semi-invalid since the influenza. Five weeks prior to admission to the Rutland State Sanatorium, cough and expectoration increased, began to lose weight and noticed fever, and blood in the sputum on several occasions. *P. E.* Lungs showed dullness over bases with many coarse mucous râles. No evidence of infiltration over parenchyma of lungs. Rest of examination essentially negative. Temperature varied from 97° F. to 100° F. *Laboratory Reports:* Negative sputum on nineteen examinations. Sputum smears showed: gram positive cocci, gram negative diplococci. Cultures of sputum: Staphylococci, streptococci, gram negative diplococci (?m. catarrhalis). *X-ray report:* "Apices clear; from sixth rib downward on the right are many white lines of fibrosis; all linear markings, especially at the bases, are thickened. Bony thorax at bases narrowed; upper thorax expanded. *Impression:* Non tuberculous lesion, probably due to an influenzal infection of long standing." *Progress Notes:* Raised large quantities of sputum at all times. *Discussion:* The history was atypical for tuberculosis, and suggestive of bronchiectasis. No evidence of lung infiltration. Signs were basilar. There were large quantities of sputum negative for the tubercle bacillus. The X-rays were negative for tuberculosis. *Discharge Diagnosis:* "Bronchiectasis".

CASE IV. Male, age 49, carpenter. *F. H.* Negative. *P. H.* Measles, whooping cough and scarlet fever. *P. I.* Influenza one year ago, which left him coughing and weak. Eight months ago cough increased, and he began to raise about four ounces of foul sputum each day. At the same time weakness increased, he began to have chills and night sweats. One month ago began to lose weight rapidly, fever and bloody sputum set in, and began to have attacks of diarrhea. *P. E.* Entered sanatorium in a weakened condition; marked orthopnea and cyanosis. Persistent explosive cough, accompanied by profuse, foul expectoration. Marked clubbing of fingers. Chest: barrel shape. Heart: action poor and rapid; sounds feeble and distant. Blood pressure —90 systolic, 60 diastolic. Lungs: upper lung fields showed no parenchymatous changes; dullness and pure bronchial breathing over the right base; many mucous râles over both bases. *Laboratory reports:* Blood agar cultures of sputum; staphylococcus albus, streptococcus anhemolyticus, and a very minute gram negative bacillus with cultural and staining characteristics of the bacillus of influenza. Blood showed a marked secondary anemia. Tubercle bacilli not found on sixteen consecutive smear examinations.

X-ray Report: "Opaque shadow right base consistent with abscess". *Discussion:* The case suggested a non-tuberculous suppurative lesion. There were symptoms and signs of abscess. The sputum showed a mixed infection, one organism resembling the bacillus influenzae. The X-rays showed abscess. The man was desperately ill, and artificial pneumothorax, to collapse the abscess, was undertaken for palliation alone. A free pleural cavity was found and compression of the lung was very easy but was abandoned

on account of the patient's poor condition. He showed a decline to a fatal termination. *Discharge Diagnosis:* "Lung Abscess".

CASE V. Male, age 38, travelling salesman. *F. H.* Negative. *P. H.* Typhoid fever at sixteen years, ill three weeks. Chancre at twenty-nine. Recurring cough, hemoptysis, and "rheumatism" of nine years' duration. *P. I.* Felt well up to two years ago; had since been "winded" and this condition was getting worse. Persistent cough past nine months accompanied by mucoid sputum mixed with blood. No loss of weight. *P. E.* Well developed and well nourished. Integument on upper lip showed several oozing lesions (? late secondary specific lesions). Chest: thick; no abnormal pulsations. Heart: action rapid, sounds weak and distant, no murmurs, no dullness over upper mediastinum. Lungs: emphysematous; scattering sibilant and sonorous rhonchi over left lung field; dullness and harsh breath sounds over upper half each lung field with many fine and medium râles over these areas. *Laboratory Reports:* Wassermann positive. *X-ray report:* "Aortic arch abnormally broadened. Numerous calcified areas. *Impression:* Aneurism and tuberculosis". *Progress notes:* Lesions on lip and "rheumatism" cleared up following medication with saturated solution K. I. Wassermann negative after three treatments with salvarsan; the patient left the sanatorium after the last treatment. *Discussion:* With the history of a primary specific lesion and symptoms of cough, dyspnea and hemoptysis, aneurism was suggested. The X-ray pointed to tuberculosis as well as aneurism. However, in spite of the abnormal signs in the upper lobes consistent with a tuberculous process, the good physical condition of the patient, and repeatedly negative sputum did not quite fit with a picture of clinical tuberculosis. Differential diagnosis rested between aneurism, syphilitic lung, and tuberculosis as a remote third. The presence of tuberculosis was at all times a matter of speculation. Clinically we were dealing with a case of syphilis complicated by lung changes compatible with a syphilitic fibrosis. *Discharge Diagnosis:* "Syphilis and ? syphilitic lung."

CASE VI. Male, age 53, laborer. *F. H.* Negative. *P. H.* Unknown. *P. I.* Began two years ago with loss of weight, and pain in left chest, at the base in front. Soon after he began to cough, raising small amounts of thick, yellow-greenish sputum often mixed with blood. Raised about two ounces of blood two weeks prior to entering the sanatorium. *P. E.* Marked emaciation. Cyanotic. No dyspnea. Numerous hard, movable masses in anterior triangles of neck. Heart: no abnormalities noted. Lungs: marked dullness over an area corresponding to the upper lobe left lung with many fine râles over this area. *Laboratory reports:* No tubercle bacilli found on sixteen consecutive smears of sputum. Guinea pig negative for tuberculosis. Wassermann negative. *X-ray report:* "Shadow of uniform density on left from sixth rib upwards. *Impression:* Abscess." A film one month later showed an extension of the process. *Pathological report on biopsied glands of neck:* "Carcinoma". *Discussion:* Differential diagnosis in this case rested between neoplasm, abscess and tuberculosis as a distant third. This case illustrates how easy it is to fall into the error of considering upper lobe pathology in the presence of cardinal symptomatology as due to tuberculosis. The man died two months after admission. *Discharge Diagnosis:* "Neoplasm of lung".

SUMMARY

1. The numerous pulmonary lesions have a marked similarity and resemble tuberculosis very closely.
2. By a process of elimination a diagnosis of tuberculosis can be made in the great majority of cases with a very small margin of error.
3. Differential diagnosis in pulmonary diseases confers a great burden of responsibility upon the physician in general and the specialist in particular.

BIBLIOGRAPHY

1 Hedblom, Carl A.: "The Diagnosis and Treatment of Bronchiectasis". Journal A. M. A., Vol. 89, No. 17, Oct. 22, 1927.
2 Atkinson, D. W.: "The Non-Tuberculous Pulmonary Fibroses". Am. J. Med. Sc., Nov., 1925, CLXX, 693.
3 Silicosis: "Special Bulletin", Bureau of Industrial Hygiene, State of New York, 1925.
4 Lechty, John A., Wright, F. K., and Baumgarten, E. A.: "Primary Cancer of Lungs". Journal A. M. A., Vol. 87, No. 13, July 17, 1926.
5 Ritvo, Max, and Clifford, Randall: "The Value of X-rays in Pulmonary Diagnosis". Boston M. and S. Journal, May 15, 1924.
6 DaCosta, John C., Jr.: "Physical Diagnosis". W. B. Saunders Co., 3rd Edition, 1916.
7 Castellani, A.: "Fungus Diseases of the Lungs that Simulate Tuberculosis". Read before the National Tuberc. Ass'n., Indianapolis, Ind., May, 1927.
8 Mullin, W. V.: "Relation of Paranasal Sinus Infection to Diseases of the Lower Respiratory Tract". Journal A. M. A., Vol. 87, No. 10, Sept. 4, 1927.
9 Voorsanger, W. D., and Firestone, F.: "Undiagnosed Cough". Read before the National Tuberc. Ass'n., Indianapolis, Ind., May, 1927.
10 Dunham, K., and Norton, V.: "Basal Tuberculosis". Journal A. M. A., Vol. 89, No. 19, Nov. 5, 1927.

THE INTERPRETATION OF APICAL RALES IN PULMONARY TUBERCULOSIS

BY RANDALL CLIFFORD, M.D.*

IN the minds of many physicians, the presence of persistent fine apical râles is considered sufficient evidence to warrant making a diagnosis of activity in pulmonary tuberculosis. The prevalence of this opinion will be realized when one considers, for example, that during the World War, in the examination of the National Army for tuberculosis, before going overseas, "the detection of persistent râles at or near the apex of the lungs was considered one of, if not *the* physical sign which called for rejection, although the soldier might have been in good physical condition otherwise, and with no evidence of general infection."[1]

That the interpretation of the clinical significance of persistent fine apical râles in cases of pulmonary tuberculosis is not, however, as simple a matter as the above would suggest has been clearly brought home to me during the past four years of work in the Pulmonary Clinic of the Massachusetts General Hospital. There has been a striking group of patients who have

*For record and address of author see "This Week's Issue," page 965.

come into the Clinic with a history of having had pulmonary tuberculosis of many years' duration. They present no evidence of any clinically active tuberculosis, are able to work without detriment to their health, and the X-ray picture is that of an old healed lesion; yet on repeated examinations of these cases definite fine persistent râles are heard at one or both apices, the character of these râles being indistinguishable from those accompanying an active process. In following these cases over long periods of time, during which on reëxamination these râles persisted, it became apparent that the râles were not in themselves evidence of active tuberculosis, and that, although similar in character to those accompanying active lesions, their method of production must be different and capable of some other explanation.

The generally accepted explanation for the production of the fine apical râles associated with pulmonary tuberculosis has been that they were vibrations caused by the currents of air passing along the respiratory passages to the air cells and meeting partial resistance, the resistance being that of fluid, mucus, or pus, the exudation being based on the inflammatory reaction of the tubercle. Therefore, as it was thought that a definite fluid medium was necessary for the production of these râles, some inflammatory process must be present to cause this fluid; and on the assumption that all râles are based on an inflammatory reaction, it was believed that fine râles heard at an apex in a case of tuberculosis must mean active disease. This, however, is in my opinion a wrong interpretation.

Very little has been written relative to the significance and method of production of these fine crepitant râles heard at the apices of the lungs in cases of healed tuberculosis.

Ringer[2], in describing the clinical criteria of activity in pulmonary tuberculosis, states that "the moist râle has ever been the one that is held to betoken activity. It matters not whether it be a fine or a coarse moist râle. . . . None the less, it is an indisputable fact that moist râles, not sibilations or large rhonchi, but true moist râles, denote an active process." Other writers, however, take issue with this view.

Riviere[3] mentions having had many cases under observation which had been long arrested but where crepitations remained. In some cases they were but the dry crackles of expanding lung, but in others the crepitations were somewhat moist and were due, he felt, to dilated bronchioles over a contracting fibroid area.

Amberson[4], in studying the healing of pulmonary deposits, finds "a surprising behavior of râles. Other signs, such as adventitious breath and voice sounds, usually change toward the normal, as one would expect, with the disappearance of pulmonary condensations, but often the distribution of râles remains as it was originally. Sometimes râles decrease, to correspond with the clearing of roentgenograms, but sometimes they persist over extensive pulmonary areas from which absorption of infiltration has occurred, and in which the roentgenographic markings are within the accepted normal limits." He gives a most interesting case in which râles persisted for more than five years after healing was well established, with a gradual clearing of the X-ray picture; they were definitely moist intrapulmonary râles, elicited by cough and at times heard during quiet breathing. In discussing an explanation for these râles, he is of the opinion that the matter is very problematical, but feels that they may be due to catarrhal bronchiolitis or to a slight bronchiolectasis, as suggested by Riviere, or to "some mechanical barrier to the blood or lymph drainage of the finer anatomic structures."

Lowenhjelm[5] remarks that at necropsy of persons with healed tuberculous processes, dying from other causes, more attention should be paid to the history of persisting râles. He describes three cases in which the clinical cure had been doubted on account of râles continuing to be heard, but in which necropsy confirmed the complete cure of the old pulmonary lesion. He found in one case that the lumen of the bronchi was unusually large and the sclerous apex was clogged with desquamated epithelium, thus providing the physical conditions for the râle production. He declares that we are inclined to attach too much importance to persisting râles.

Bruns[6] believes that the class of râles elicited by expiratory cough and inspiration and commonly regarded as an important sign of chronic parenchymatous tuberculous lesions are not, as generally believed, produced by moisture or secretions in the terminal bronchioles, but that "their production seems to depend on a certain degree of collapse of the secondary lung lobules; furthermore, that these râles, while usually regarded as typical of tuberculosis, may be present without active lesions, and therefore they cannot be accepted as conclusive evidence of active or even manifest tuberculosis unless supported by other findings." In describing these râles, Bruns states that they are small or medium but uniform in size and occur in showers at the same phase of respiration, and are encountered in all types of tuberculosis. In concluding, he remarks that "the significance of these râles as a criterion of manifest tuberculosis and the character of the lesions will depend on other clinical findings—history, symptoms, sputum, roentgen-ray examination. A period of observation may be necessary in order to arrive at a definite diagnosis."

We cannot say, at the present time, whether the true physiology of persistent apical râles unassociated with any symptoms is based upon fibrosis, bronchial obstruction, or some other factor. It would seem to me that the manner of production of these fine râles occurring in cases of long-standing tuberculosis must be different from those accompanying active lesions.

When the disease is active, we can logically assume that the moisture in the bronchial tubes giving rise to the râles is secondary to some inflammatory process. On the other hand, where healing has taken place and no acute inflammatory condition exists, some explanation other than moisture in the bronchial tubes must be sought. The primary clinical problem, however, is not so much how these râles are produced as the determination of whether or not they should necessarily be considered proof of activity in the absence of other factors. It is my opinion that persistent fine apical râles alone, in the absence of symptoms, are not sufficient to warrant making a diagnosis of activity in pulmonary tuberculosis. The following cases, seen in the Pulmonary Clinic of the Massachusetts General Hospital, illustrate this point of view.

CASE I. A man of forty-five entered the Pulmonary Clinic in 1927 for examination of his lungs. He gave a history of having been examined in the Out-Patient Department in 1920, when a diagnosis of pulmonary tuberculosis was made and he was sent to a sanatorium, where he stayed for a year and left against advice. He had then gone to work and had been working ever since without any apparent symptoms. Before entering the Clinic in 1927, he had seen a doctor, who, finding râles at the right apex, thought that he had active disease and advised that he return to the sanatorium. The examination in the Pulmonary Clinic showed him symptom-free, but with definite râles at the right apex with and without cough; the X-ray showed practically no change from that taken in 1920. After following his case for several months and finding that he was able to work without any symptoms in spite of these râles heard at each examination, I told him that I thought the disease was not active and advised that he continue his work which he had done for the past six years without any ill effect. The case of this man, entirely without symptoms and showing no progression of the lesion by X-ray in seven years, who yet because of fine apical râles was told that he had active disease and needed sanatorium treatment, illustrates very clearly the value of following such a case closely before deciding whether or not the râles indicate active disease.

CASE II. A woman of fifty-two entered the Out-Patient Department in 1913. At that time, dullness and fine râles were found at the right apex, the sputum was positive, and a definite diagnosis of active pulmonary tuberculosis was made. When she returned in 1919, dullness and râles were still found at the right apex, but the disease was no longer considered active. In 1925, she came into the Pulmonary Clinic, and again dullness and râles were found at the right apex, the X-ray showing a diffuse bronchial and peribronchial process affecting both lungs, with evidence of fibroid changes at the right apex; that same year she had a gall-bladder operation without any apparent setback. In 1927, she again came into the Clinic, giving a history of essentially no constitutional symptoms; in view of the absence of symptoms and the long duration of the physical signs I believed that the râles heard at the right apex, although indistinguishable from those which accompany an active process, were based on fibrosis and were not indicative of activity, and my diagnosis was chronic fibroid phthisis. This is a case in which for fourteen years fine râles at the right apex were heard at varying intervals by different observers, yet the patient apparently continued in perfectly good health and without any symptoms.

CASE III. A woman of thirty-one entered the Out-Patient Department in 1923. She had a slight cough with apparently no other symptoms; her lungs showed dullness at the right apex to the mid scapula with increased breath sounds and fine râles after cough; the X-ray showed mottled dullness at both apices, consistent with tuberculosis. She gave a very strong family history of tuberculosis, and had been for four months in a sanatorium, which she had left against advice. She entered the Pulmonary Clinic, where she made eleven visits between November, 1924, and October, 1926; at each examination, definite fine râles were heard at the right apex. The X-ray taken in 1924 was similar to that taken in 1923, but showed some calcified areas and mottling at both apices. In 1925 she became pregnant and was advised to go to the Lying-In Hospital. She next appeared at the Clinic in 1926, saying that her child had been born seven months previous; she was feeling extremely well, but examination still showed the same râles at the right apex. The fact that she went through a pregnancy without any change in her lung condition is good evidence that the râles in this case were not associated with an active lesion.

CASE IV. A woman of forty entered the hospital in May, 1923, when a diagnosis of inactive pulmonary tuberculosis was made. She gave a history of having been at a sanatorium fourteen years ago; the physical examination showed a few fine râles at the right apex after cough, with slight dullness and increased breath sounds; and the X-ray showed mottling at the right apex. In October, 1925, a doctor saw her and thought that she ought to go back to the sanatorium. She came to the Pulmonary Clinic for the first time in December, 1925; at this time the X-ray showed evidence of fibrosis at the right apex and many small calcified areas which probably represented an old healed tuberculous lesion. She has since made numerous visits to the Clinic, and at all times definite fine râles have been heard at the right apex, although she has been working without any undue fatigue and has gained in weight. This case emphasizes the difficulty in interpreting the significance of apical râles, for unless one had followed the case for a certain length of time and had carefully noted the absence of any symptoms, one might have been inclined to consider the disease as active on the basis of the physical signs alone; in fact, we note that although she has apparently been perfectly well ever since leaving the sanatorium, yet on the basis of râles at the right apex a doctor who saw her in 1925 felt that the disease was active and advised that she return to the sanatorium.

CASE V. A woman of twenty-five entered the Clinic on September 2, 1925, for the question of her fitness for an operation for prolapse of the uterus. Three years earlier she had been seen in the Out-Patient Department, when the physical examination showed dullness at both apices, increased voice and breath sounds, and fine râles at the left apex after cough; the X-ray showed evidence of calcification in the peribronchial structure on the left side, and considerable fibrosis and retraction of the mediastinal contents toward the left; a diagnosis of moderately advanced tuberculosis was made and she was advised to go to a sanatorium. At the time of her entrance to the Pulmonary Clinic in 1925, she gave a history of having raised a little blood about three years previous; otherwise, her history was essentially negative, and the X-ray showed no appreciable change from the one taken in 1922. The examination showed dullness at the left apex, front and back, with numer-

ous fine râles; and in spite of no elevation of temperature or toxemia, I advised against an operation at that time on the basis of these râles. The operation was accordingly postponed. Nine months later she came in again, showing practically the same signs, and with no change in the X-ray. I changed my opinion in regard to the operation, feeling that these râles at the left apex, having persisted since 1922, did not, in the absence of toxemia, mean an active tuberculous process, and that I therefore was not justified in advising against an operation on this evidence alone. The patient was accordingly operated upon and suffered no ill effect or reactivation of the process.

CASE VI. A plumber of thirty-eight first entered the hospital in 1920 when evidence of an old right apical involvement was found, but there were no signs of activity. The X-ray showed mottled dullness extending from both lung roots toward the apices; on the right, these changes reached the periphery of the lung. The findings were typical of tuberculosis, and in 1921 he went to a sanatorium. In 1925 he entered the Pulmonary Clinic, saying that he had been working for the past six months as a plumber and had been feeling well. The physical examination showed dullness at the right apex with a few fine râles after cough. The sputum examination for tubercle bacilli was negative. He has been followed in the Pulmonary Clinic since 1925, being last examined on March 30, 1927, and on all of these examinations definite fine râles have been heard at the right apex. He has gained five pounds in weight, has had no night sweats or other symptoms, and the 1927 X-ray plate shows the same picture as did that taken seven years ago in 1920. The presence of persistent fine râles occurring on examination for the past two years, and in all probability since 1920, brings up the question as to whether or not this case is active. In my opinion, the facts that he has been working as a plumber without physical detriment, that the X-rays taken in 1925 and 1927 show no marked changes from those taken in 1920, and that constitutional symptoms are absent, are sufficient evidence to warrant diagnosing this case as quiescent, even in the presence of persistent fine râles at the right apex.

I feel sure that there are many tuberculous patients being treated as confirmed invalids merely because of the misinterpretation of persistent fine râles at an apex. There are physical signs in the chest which may be of the greatest significance in one instance and may mean very little in another, depending upon the ability or inability of the patient to carry on his normal activities in life. Dr. E. O. Otis[7] speaks of this as follows: "It is true that in the greater number of cases when râles are present at one or the other apex there are co-existing symptoms of a general infection, and the râles are the local evidence of the constitutional disturbance. It is the toxaemic symptoms that are the indication of the active disease, not the râles. When, therefore, all evidence of tuberculous toxaemia has disappeared, but the râles remain, they then indicate only what has happened, and one should bear this in mind in forming an opinion upon the progress of a case under treatment. Of course one may say that as long as râles remain, the disease is only quiescent and toxaemic symptoms may appear at any time when the patient is under stress. This, indeed, may happen, but on the other hand it may not, and the test is the continued physical activity of the patient, and when this takes place without detriment, the disease may be considered as permanently arrested."

Fine râles at an apex in long-standing cases of pulmonary tuberculosis do not *alone* prove activity or the reactivation of an old process. A man may have a healed scar in his lung which never has caused him inconvenience and probably never will; and to deprive such a man of his occupation and disrupt his family life on the sole finding of fine râles at an apex, unaccompanied by symptoms, may have consequences as serious as might a failure to recognize the significance of such râles in an early case.

CONCLUSION

1. Symptoms are more important than physical signs in judging activity in pulmonary tuberculosis.
2. Fine apical râles may persist for long periods of time in cases of healed tuberculosis.
3. I believe that fine apical râles are not necessarily dependent for their production on moisture in the bronchi, secondary to an inflammatory reaction in the lung, but are in many instances due to collapse of the lung lobules.
4. Persistent fine apical râles alone, in the absence of symptoms, are not sufficient to warrant making a diagnosis of activity in pulmonary tuberculosis.

REFERENCES

1 "Some Misleading Beliefs Regarding Pulmonary Tuberculosis," E. O. Otis, Military Surgeon, February, 1921.
2 "The Clinical Criteria of Activity in Pulmonary Tuberculosis," P. H. Ringer, Am. J. Med. Sc., 165:742, 1923.
3 "Damaged Lungs and Bronchiectasis," C. Riviere, Brit. M. J., 1923, No. 3239, p. 141.
4 "The Absorption of Pulmonary Deposits," J. B. Amberson, Jr., Am. Rev. Tuberc., 10:227-248, Nov., 1924.
5 "Râles After Clinical Healing," C. Lowenhjelm, Acta Medica Scan., Aug. 23, 1921, 55, No. 4.
6 "Parenchymal Râles in Pulmonary Tuberculosis," E. H. Bruns, Jour. A. M. A., Sept. 5, 1925.
7 "Some Misleading Beliefs Regarding Pulmonary Tuberculosis," E. O. Otis, Military Surgeon, February, 1921.

MASSACHUSETTS TUBERCULOSIS LEAGUE

FUNDAMENTALS IN TUBERCULOSIS WORK WITH RESPECT TO VOLUNTARY UNOFFICIAL ASSOCIATIONS*

BY GEORGE J. NELBACH†

THE subject of this paper will be discussed under two general heads: (1) fundamentals with respect to policies; (2) fundamentals in regard to programs of work.

One of the fundamental policies with respect to voluntary tuberculosis associations is that these organizations should seek to promote and secure mutual understanding, friendly appreciation and coöperation with the constituted public health authorities, state and local. To this end there needs to be a due recognition by each group of the other's basic character and organization.

First, as to the state and local health authorities. They are the agencies of government. They represent all the people. They are created to carry out the will of the people, as expressed in statutes, charters, codes, ordinances, rules and regulations. They spend public moneys which all the people provide directly or indirectly through the taxes that are imposed by the people's law-making and tax-levying representatives in government.

Under the system of self-government prevailing in our country, the people have established certain limitations and checks upon the powers and duties of those whom they have selected as their agents in public office, and the official health authorities, whether state or local, are subject to these checks and limitations. For example, their tenure in office is fixed usually for a specified term—generally two years, some times three, occasionally four years, rarely more than that, though the trend is in that direction. Whether they shall be re-elected or re-appointed to office is subject, directly or indirectly, to the will of the people, and sometimes the public is capricious in expressing its will. This insecurity as to tenure in office deters many from entering into official public health work as a career. Powers and duties of public health officials are defined and prescribed by state statutes or city charters, and beyond these provisions they cannot go. They can only spend the moneys that are appropriated to them for the particular purposes that are specified and not otherwise. They are subject to minute regulations as to budget making, to manner of expenditure of public funds, and as to accounting therefor.

Appointments to subordinate positions are usually subject to civil service rules, which may possibly be interpreted or administered by uninterested or unsympathetic civil service officers. It occasionally happens that a particular health administration inherits from a preceding one a group of subordinates of an inferior type, appointed when a lower standard of duty and work were required, and who are protected by civil service regulation. Such subordinates can only be removed on charges and after a hearing. The personnel of such official health agencies cannot for the most part be "hired and fired" like those of the voluntary, unofficial tuberculosis agencies.

Such limitations as these often inhibit the initiative and thwart the enterprise of public health officials, state and local, and condition the extent and character of their work. It is well for the voluntary health agencies to recognize these handicaps and to make allowance for them. For the past twelve years experience everywhere in this country indicates that a voluntary health organization, parallel with the official health authority, is an indispensable means for building up modern official health work and keeping it at a higher standard. Theoretically, the will of the people is expressed in laws and public institutions. In matters relating to sanitation and public health, however, there is little public opinion because the mass of the people are largely without much knowledge of the subject. Under these circumstances a disinterested voluntary organization, expressing the consensus of opinion of physicians and others already interested in public health, is an invaluable nucleus for the development of a wider public knowledge and concern; and for carrying on from time to time some of the newer activities in public health until their practicability and usefulness are demonstrated, public interest in them aroused, and a desire on the part of the public to provide for their support by taxation is developed.

The voluntary health agencies have considerable freedom of action, including, among other things, the raising of money, the spending of money and the employment and dismissal of members of their staffs. They also have considerable elasticity as to function; it is relatively easy for them to change their programs of work; their functions are not minutely prescribed by statute. In fact only such voluntary health agencies as are incorporated have their functions prescribed legally, and as to these agencies, wide latitude is usually permitted them in the interpretation and exercise of these functions.

The voluntary health organization helps to

*Remarks made before the Annual Meeting of the Massachusetts Tuberculosis League, Springfield, Mass., April 30, 1928.

†For record and address of author see "This Week's Issue," page 965.

reduce the danger of serious retrogression when changes occur in the personnel of the public health board or department, such as seem inevitable from time to time under present political conditions. It serves as an organ of continuity of objectives and standards, by creating a continually informed public opinion which demands efficient results from whatever administration or officials occupying the executive positions. The voluntary health organization gives a type of publicity to the work of the official health board or department that the latter itself cannot obtain without embarrassment. It interprets the board's or department's work to the people as citizens and taxpayers; it builds up for it an increasing fund of good will and favorable sentiment, and thereby wins for the official board or department increased public recognition and support.

The voluntary, unofficial tuberculosis associations are for the most part made up of sincere, sensible, disinterested, public spirited physicians and lay persons, bankers, industrialists, merchants, trade union representatives, club women, persons who are genuinely concerned about the advancement of the public health and welfare. In the earlier years of the tuberculosis movement it was not uncommon to find public health officials, particularly the more smug and self-satisfied kind, looking upon the voluntary, unofficial tuberculosis associations as made up of "long haired men and short haired women", impractical, visionary enthusiasts, always poking their noses into somebody else's business, vain—glorious seekers of place and publicity for themselves. But this concept almost everywhere has passed away in the face of the practical demonstration by the voluntary organizations of the value of their coöperative services to the official health authorities.

Certain expedients have been found useful for facilitating and maintaining effective working relations between official and non-official health agencies. Among them are the following:

(a) The voluntary health agency invites the official health board or department to designate its chief executive officer to become a member ex-officio of the board of directors or executive committee of the voluntary health association.

(b) The official health board or department persuades the governor of the state or the mayor of the city, as the case may be, to appoint a leading member of the voluntary health association to membership on the advisory board which nowadays almost every official health agency has.

(c) The voluntary health agency submits its tentative program of work to the official agency for information and suggestions, though not for approval.

(d) The official health agency endorses the voluntary health organization's program of work, and its campaign for the raising of funds for the support and execution of its program.

(e) The voluntary health organization tenders to the official health board or department its good offices in creating such public sentiment as will help the official health board or department to secure necessary legislation or increased appropriations of public moneys for its work.

(f) In addition to reciprocal ex-officio representation mentioned in (a) above, frequent conferences are held between heads of divisions or bureau chiefs who are engaged in similar phases of public health work. This tends to improve team play all along the line, and to head off possible troubles that might develop between subordinates of the two agencies.

The official health agency is from every point of view the responsible head of the work. It has been set up by the constituted authorities and is supported by the taxes levied on the citizens of the community to discharge the duty of protecting the health of the general public. It has no choice as to the kind or degree of responsibility it will assume. It is all its responsibility. The constituted legislative authorities have seen to that. It is, therefore, plain that the place in the picture of the voluntary unofficial agency is that of an aide and supporter of the official health agency in every move that the latter makes for the public's benefit. The official health agency is the spear head of the drive; behind it is the informed, earnest, active group of public-spirited men and women, physicians and lay persons, who constitute the voluntary health agency and who represent enlightened public opinion.

The ideal combination, in my opinion, is that of the official health agency and the non-official one working in close coöperation, but with a clear distinction between them. If the non-official agency is in any very material respect closely bound up with the official health agency —if for example, one or more of the chief executive officers of the official health agency are also among the principal officers of the voluntary health agency, as well as on the staff of the official health agency, the voluntary health organization loses its identity in the public mind, and in effect becomes a mere adjunct to the official board or department. Such a close affiliation deprives the voluntary health organization of its independent, disinterested character; its initiative and freedom of action are stifled. It really deprives the official health board or department of the peculiarly unique and valuable assistance that the voluntary health organization can render to the official health agency.

Another fundamental policy to be adopted by the voluntary unofficial tuberculosis association is the securing of a friendly understanding, good will and coöperation on the part of the medical profession. The variety and extent of anti-tuberculosis measures have increased greatly in recent years, in common with other lines of preventive health work. Naturally the points of contact between anti-tuberculosis agencies and

medical practitioners and medical societies have increased greatly. Sometimes these points of contact have become points of friction. The more intensive and comprehensive the health activities, the greater has been the likelihood of friction.

Sometimes physicians have complained that tuberculosis agencies offer medical examinations and services free of charge at clinics which the patients should receive through their physicians on a professional basis; that tuberculosis work has not always been wisely planned, nor conducted on sound scientific principles; and that tuberculosis work along with other health services is leading up to state medicine.

On the other hand, health and tuberculosis workers here and there have complained that physicians have been holding aloof from their natural place in health work; that they have condemned health activities without taking the trouble to understand them and on inaccurate information; that preventive tuberculosis work should not be put on the same economic basis as the treatment of the indigent sick, and that instead of leaning toward state medicine the progressive development of tuberculosis and other activities tends to lessen the need for any public system of medical practice.

The situation, it seems to me, is one calling for patient, thoughtful, forbearing consideration of the whole general subject. Progress will not be made by strife and contention between the medical profession and public health work. More light and less heat is what is called for.

In New York State the conviction has gained ground that the difficulties may be due to the fact that the relations of modern health activities to the medical profession present a relatively new subject to which no one has as yet given serious study. Two years ago it seemed worthwhile to a group of physicians and laymen to give very detailed consideration to just what part the medical profession in a given locality as an organized group and as individuals can play, actually and practically in the health activities of that community; and as to what procedures and relationships between health and medical agencies would be likely to lead to a mutual understanding, based on actual and detailed information. It was felt that if there were underlying conflicts of interest, such mutual consideration would at least bring them to light and narrow the discussion to actual realities instead of vague generalities and loose terms. A series of informal conferences was held, and these were succeeded by a series of formal ones between specially appointed committees of the State Medical Society and of our State Committee on Tuberculosis and Public Health.

Of course, there were plenty of differences of opinion among the two groups as various steps were taken up, but seldom were the State Medical Society's representatives on one side of a particular question, and all those of our State Committee on Tuberculosis and Public Health on the other. The discussion was entered into with good will on the part of all, with an earnest effort to learn what the facts really were, and to arrive not simply at a modus vivendi for the time being, but at a well considered and statesmanlike handling of the subject. The result was a unanimous report dealing with general considerations as to the relations between medical organizations, and voluntary tuberculosis and health agencies, and suggestions in detail as to the operation of various health activities. The report was submitted informally for suggestions and comments to the various state authorities concerned; the State Commissioner of Health, the State Commissioner of Mental Hygiene, and the head of the School of Health Service in the State Department of Education. Some valuable suggestions were received and the comment was entirely favorable. In May 1927 the report was adopted by the parent bodies of both committees—the State Medical Society and the State Charities Aid Association. The report was printed in full in the *State Medical Journal*, issue of July 15, 1927, the official publication of the State Medical Society, and in the *S. C. A. A. News*, issue of July 1927, the Bulletin of the State Charities Aid Association.

The report, of course, is too long to be set forth within the compass of this paper. However, it may be useful to give at this point the suggestions that were arrived at as to relations that should in our opinion be set up between medical organizations and voluntary tuberculosis associations.

The following suggestions were made to the voluntary tuberculosis associations:

(a) Invite the County Medical Society to designate a certain number of its members, say two, to become members ex-officio of the tuberculosis association's board of directors or executive committee, if there be one.

(b) Lay its annual program of activities before the County Medical Society for information and suggestions.

(c) Appoint a special committee to confer with a similar special committee appointed by the County Medical Society about matters that come up from time to time in which both groups are mutually interested.

(d) Report to the parent body of the voluntary tuberculosis association any matter as to which an accord has not been reached with the County Medical Society, in the hope that through conference between the parent body of the tuberculosis association and the State Medical Society any such matter may be negotiated and adjusted to the mutual satisfaction of all concerned.

(e) Conduct at least one open meeting each year to which the members of the County Medical Society are specially invited.

The following suggestions were made to the County Medical Societies:

(a) Accept the invitation from the voluntary tuberculosis association to designate certain of its members to serve as members ex-officio on the board of directors or executive committee of the association.

(b) Study and consider any program of activities that may be referred to it by the tuberculosis association for information and suggestions; and, if such program is approved, make a public statement to that effect, and otherwise support the program so approved.

(c) Appoint a special committee on public relations to confer from time to time with a similar committee appointed by the voluntary tuberculosis association on matters of mutual interest to both agencies.

(d) Report to the State Medical Society any matter as to which an accord has not been reached with the tuberculosis association, in the hope that through conference between the State Medical Society and the parent body of the tuberculosis association any such matter may be negotiated and adjusted to the mutual satisfaction of all concerned.

A substantial number of the county medical societies and voluntary tuberculosis associations have acted affirmatively on these suggestions, and it is believed that more of them will do so.

As to the tuberculosis program, it is fundamentally important that it should be soundly conceived. It should have a scientific foundation, and should meet the needs of a particular situation in the state or in a given locality within the state, at a particular time. Fortunately there is but little likelihood of any state tuberculosis association or similar local association going wrong, that is to say, undertaking relatively unfruitful, ineffective lines of anti-tuberculosis endeavor, if it will but follow the suggestions as to program making that have been evolved out of the experience of the past 25 years of work in this field throughout the country, and as formulated by the National Tuberculosis Association. The Board of Directors of the National Association has from time to time studied the relative value of the various lines of anti-tuberculosis work, such as, survey, education of the public, legislation, clinic, sanatorium, public health nursing, preventorium, open air school, charitable relief, post sanatorium care. Its recommendations with respect to these activities have been issued formally to the 48 State Tuberculosis Associations, and through them to their 1500 or more county or city branches. The recommendations with respect thereto are to be found year after year in the annual contract which these agencies execute among themselves with respect to the sale of the tuberculosis Christmas Seals, the device by which these Societies finance their activities.

Case Records of the Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 14181

EDEMA OF THE FEET

MEDICAL DEPARTMENT

An Irish-American farmer sixty-eight years old entered March 9 complaining of swelling of the ankles of four months' duration.

Eight years before admission the patient had an illness similar to the present one, with dyspnea and edema of the legs, lasting for a few weeks.

For a year or two he had urinated three or four times at night and had had a little difficulty in starting the stream. For a year he had not been quite so "spry" in his work as usual. He had, however, continued to do a great deal of work every day until four months before admission. Then he began to have dyspnea on exertion, orthopnea, swelling of the ankles and high colored, often reddish urine. He continued to do some work until three weeks before admission. He thought that exercise as well as a night's rest caused the edema to go down. When it was marked he had pain in his shins and calves, severe at times, especially at night. He had grown more and more dyspneic and in the past month had begun to have attacks of nocturnal dyspnea waking him from a sound sleep, lasting about an hour and forcing him to sit up to breathe. For six weeks he had had cough, chiefly at night, with a quarter of a glassful of whitish sputum daily. For four weeks he had been taking digitalis. Two weeks ago he had nausea and vomiting for two days, due his doctor said to overdosage of digitalis.

His mother had heart disease and rheumatism for the last twelve years of her life. She died of pneumonia.

Clinical examination showed a man sitting up in bed with slightly rapid but not labored breathing. Skin loose; apparent loss of weight. Small deep bilateral supraclavicular glands. Bean sized inguinal glands. Barrel chest. Moist râles at both bases. Heart fibrillating at a slow rate. Forceful apex impulse seen and felt in the sixth space. Left border of dullness 15 centimeters to the left of midsternum, 4.5 centimeters outside the midclavicular line, right border 3.5 centimeters, supracardiac dullness 8 centimeters. Sounds of good quality. A soft systolic murmur all over the precordium, best heard over the tricuspid area. Blood pressure: right 142 to 128/72; left 145 to 125/75. Pulses normal. Artery walls palpable and tortuous. Liver dullness from the fifth rib to 4.5 centimeters below the costal margin; edge palpable. Resistance to palpation, probably liver, 6 centimeters below the costal margin, slightly tender. Marked pitting edema of the legs from the thighs down. Skin over the right instep and lower leg red, wrinkled and in places broken. Slight edema of the prepuce. A red area over the tip of the sacrum—non-patent fistula. Prostate large, firm, symmetrical, not tender. Pupils normal. Fundi: right disc margin indistinct, left showed a white lateral crescent; vessels tortuous. Reflexes normal.

Amount of urine not recorded, specific gravity 1.022, cloudy at both of two examinations, a large trace of albumin at both, sediment negative. Blood normal. Wassermann not recorded. Non-protein nitrogen 43 milligrams. Icteric index 5.

Temperature 97.1° to 101.9°, rectal. Pulse 61 to 102. Respirations 25 to 14.

March 12 the blood pressure was 190/105. The following day the heart rate was about 85. At the apex was a fairly loud short systolic murmur, transmitted toward the axilla. The first sound here was of fair quality, the second sound normal. In diastole here there was a middiastolic third sound. There was no systolic at the base and no diastolic there or along the left border of the sternum. On March 13 there were small shallow ulcerated areas on the third and fourth right toes, painful and tender, partly covered with white dead skin. The entire right foot and lower leg were dusky red and slightly colder than the left. The dorsalis pedis pulsation was felt in the left but not in the right foot. On the left a vessel could be felt to pulsate anterior to the external malleolus. A surgical consultant advised no further treatment.

The patient was so noisy that he had to be transferred to a private room. Plans were made for his care at home, though it was questioned whether he was able to stand the journey. It was thought that possibly digitalis might be responsible for his dementia. He became more and more unmanageable. He made response to morphia, but only limited use of the drug was possible because of his respirations. In spite of it he overpowered his attendants several times and got out of bed. March 18 in such an excursion he became hyperactive and fell suddenly to the floor, cyanotic and breathing rapidly. Soon after being put back to bed he died.

DISCUSSION

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

1. "Exercise as well as a night's rest caused the edema to go down." That is a thing that we are struck with sometimes in taking his-

tories of cardiac patients. Ordinarily the rule is the other way; exercise makes the edema come. There are some patients—and I have not been able to distinguish of what type they are—who walk off edema instead of walking it on. That seems to be the case here. Ordinarily when the patient gets up in the morning the edema comes and when he goes to bed it goes off. But sometimes a patient can get rid of edema by using his muscles.

2. "Pain in his shins and calves." I suppose that is from the same cause that makes edematous parts so tender. When you are feeling along the shins you will find that every now and then the patient winces. There is tenderness there more than elsewhere. I suppose it is due to some physical or chemical change in the nerve endings from the presence of the fluid.

3. As the mother's heart disease and rheumatism occurred in the last twelve years of her life we probably can infer that it was not the rheumatism that produces heart disease but the stiffness and soreness about the joints which old people have, and which have no connection with heart disease.

NOTES ON THE PHYSICAL EXAMINATION

1. The right and left blood pressures were identical. Those differences are less than any we can measure with any blood pressure machine that I know. I suppose that they were thinking of the possibility of aneurysm. That is the only condition in which we get big differences of blood pressure on the two sides.

2. The red area over the tip of the sacrum is a danger signal for a bedsore. If that is not very carefully taken care of, shielded by "doughnuts" and things of that kind, you do not escape the bedsore. Where else should we look for red areas?

STUDENTS: On the ankles—heels—scapulae—great trochanter of the femur.

3. DR. CABOT: There was an opening into which they could put a probe, but into which they could not get far. I should say it was not a fistulous opening, but a pilonidal sinus.

4. What do you infer from the fundi?

A STUDENT: Arteriosclerotic changes.

DR. CABOT: With or without hypertension.

5. The laboratory findings are all negative.

6. If we were certain that he had a third sound and not a murmur we could infer nothing. It is one of the most difficult things in ausculation to tell a third heart sound from a mid-diastolic murmur. Very few practicing physicians that I know can do it. It is quite a possible thing to teach, but it did not come up as a thing necessary to teach in our medical schools until recently. We have no positive evidence here either at the apex or anywhere else of a diastolic murmur.

7. If the man could not stand the journey home he could not stand amputation. It was considered and refused.

8. That death again is one of those incidents that you would much rather have happen in an institution than outside. If it happened outside it would certainly be said that the nurse murdered him.

DIFFERENTIAL DIAGNOSIS

From a purely medical point of view I do not think the case offers much difficulty. We have been given the facts on the heart and we have to say there is hypertrophy and dilatation. We know that hypertensive heart disease at his age is overwhelmingly more common than any other type of heart disease. Hypertensive heart disease with cardiac hypertrophy and dilatation are the fundamental things. Failure of the heart in that condition is what gives the dyspnea and the tender condition and edema of the legs. We have to account for that arterial embolism which I have no doubt he had in the right leg. We have got to suppose the possibility of an intracardiac thrombus in the left ventricle. Experience shows that these arterial thrombi usually extend farther up than we expect. We have evidence only below the knee. Unless I am mistaken I think we shall find that his goes away up into the pelvis.

What can we say about the kidneys? I think they will be found to be arteriosclerotic. There is no good evidence of nephritis. There is good gravity of urine, a negative sediment, a fairly low non-protein nitrogen. I should say the kidneys ought to show general arteriosclerosis but not nephritis.

A STUDENT: Was the sugar test done?

MISS PAINTER: The urine showed no sugar.

DR. CABOT: As to his brain we will not argue, as Dr. Mallory did not get permission to examine the head.

The lungs should show general passive congestion and hydrothorax, although we have no evidence of the latter.

The liver of course will show passive congestion. Presumably there will be some fluid in the peritoneum.

A STUDENT: Do you have to suppose an arterial embolus in that leg?

DR. CABOT: No, you do not. You can have peripheral arterial thrombi with no source found. You always wonder if you have not missed something. I am right in that, am I not, Dr. Mallory?

DR. TRACY B. MALLORY: Yes, sir.

A STUDENT: Is a concentrated urine red, as it says here?

DR. CABOT: Yes, reddish.

A STUDENT: What was the leading cause of death?

DR. CABOT: Exertion in a patient with a heart having very little marginal reserve.

A STUDENT: Do you think that it is necessary to assume cerebral thrombosis or meningitis or anything of that sort?

DR. CABOT: I do not care to spend time on

that. If the head had been examined it would have been worth while to go into it. I do not believe he had meningitis. I doubt if he had anything more than the arteriosclerotic process; that could account for all the mental symptoms. Patients have to be put into insane asylums for pure cerebral arteriosclerosis.

A STUDENT: How far may renal arteriosclerosis go without being called nephritis?

DR. CABOT: As far as the pathologist allows. That is up to the individual expert. I am predicting what Dr. Mallory will say. You have to know your pathologist, however.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Arteriosclerosis, general and cerebral.
Congestive failure.

DR. RICHARD C. CABOT'S DIAGNOSIS

Hypertensive heart disease.
Hypertrophy and dilatation of the heart.
Embolism of the right leg.

ANATOMIC DIAGNOSES

1. *Primary disease.*

Cardiac infarction.

2. *Secondary or terminal lesions.*

Bronchopneumonia.
Arteriosclerosis, generalized (including pulmonary).
Passive congestion, general.

DR. MALLORY: The most important finding in the case was an immense heart weighing 840 grams,. Both cavities took part in the hypertrophy, the left however considerably more than the right. The entire apex of the ventricle was destroyed by a large infarct nearly 5 centimeters in diameter. Over this a thrombus was adherent on the endocardial surface. There was no change in the epicardial surface. A portion of this thrombus had broken loose and produced the embolus in his leg. It is also quite possible, although we have no evidence, that an embolus in the cerebrum may have been the immediate terminal cause of death.

The descending branch of the left coronary artery showed very marked sclerosis, with at one point a small calcified mass which almost completely—not quite—occluded the lumen of the vessel. There is always the possibility in these cases, of course, that a thrombus has occurred at some previous time and has become recanalized, so that in many of these cases of long-standing infarct of the heart we do not find completely occluded coronaries. The infarct here was of long standing, since marked fibrosis had occurred. The heart muscle had been for the most part absorbed, thinning the wall of the ventricle at the apex down to less than two millimeters. At this point it bulged out in the form of a small but quite definite aneurysm of the heart itself.

The lungs showed a slight degree of emphysema, and microscopically a few areas of bronchopneumonia were found, but not enough to have been of much significance in the clinical picture.

The rest of the organs showed a marked chronic passive congestion.

The kidneys showed only a moderate degree of arteriosclerosis, many changes in the larger and medium sized vessels, very few in the glomeruli themselves.

I think in answer to what Dr. Cabot was saying as to when we can call vascular changes in the kidney nephritis, there is no hard and fast rule. Every pathologist does have his own criteria. Possibly the chief point is whether there is a more or less generalized process in the glomeruli. With arteriosclerosis of the large vessels only, clusters of glomeruli supplied by the single vessels, which have become thrombosed and occluded, undergo a slow type of infarction, and we get fibrous scars. In general that type of fibrous kidney seems to produce no clinical symptoms other than possibly hypertension. When, however, one has a diffuse process affecting to a slight degree only perhaps, all the glomeruli, we have signs of renal insufficiency at one stage or another. In this kidney there was practically no change in the minute arteries, the afferent arteries of the glomeruli, and very few changes in the glomerular capillaries. It is a case in which I should feel quite confident in saying from the histologic section only that he would not have shown any true renal symptoms other than hypertension.

DR. CABOT: How far did the embolism extend in the artery?

DR. MALLORY: It was down below the thigh.

DR. CABOT: This man had a cardiac infarct. What is the ordinary symptom of cardiac infarct?

A STUDENT: Pain.

DR. CABOT: Yes; and he did not have that. It is not extremely rare to have a case of cardiac infarct without the only symptom by which we can distinguish it. If there is no pain we cannot make the diagnosis. The very large majority of cases that I have known have had pain. If this man had it he did not tell anybody, or nobody told us. Was this a matter of months?

DR. MALLORY: Yes; it was two months at the very least.

CASE 14182

NEGLECTED HEMATURIA WITH FATAL RESULT

UROLOGICAL DEPARTMENT

An Irish-American laborer sixty-seven years old came to the Emergency Ward January 10 complaining of hematuria.

A little over two years before admission he

noticed that his urine was bloody and sometimes contained clots. He had dysuria, worst at the end of urination. This had gradually increased in severity. After three months of treatment in his home town he was sent to the nearest large city, where a cystoscopy was done and three treatments given, the last one three months before admission. The first of these treatments stopped the bleeding for about three months. The other two had little effect. He had had intermittent dull suprapubic pain for the past three months, usually more intense just before urination. For ten years he had urinated once or twice at night. He now urinated ten to twenty times at night and every half hour by day, passing sometimes only a spoonful. With the onset of the hematuria the nycturia suddenly increased. Since the onset of the present illness and especially during the past three months he had had frequency. For the past few months he had had dribbling.

Three sisters died in their early twenties, probably of tuberculosis.

He had had rheumatism in his back for the past ten years. He had dyspnea on moderate exertion. He had lost ten or twenty pounds in the past few months.

Clinical examination showed a fairly well nourished man. Lungs clear. Apex impulse of the heart not seen or felt. No enlargement to percussion. Sounds of good quality. An occasional extrasystole. A loud systolic murmur over the apex. Artery walls palpable. Blood pressure 170/100. Liver could not be mapped out, probably because of gas in the colon; possible atrophy. Abdomen otherwise negative. Prostate seemed diffusely enlarged and somewhat firmer than usual. Marked tenderness. No adherence to the rectum. Pupils normal. Early cataracts. Reflexes normal.

Before operation urine normal in amount, specific gravity not recorded, a slight trace to a very slight trace of albumin, sediment grossly bloody at both of two examinations, with many leukocytes. January 15 residual 20 cubic centimeters of bloody urine with bits of whitish tissue. Renal function 60 per cent. Blood not recorded. Wassermann negative. Non-protein nitrogen 33 to 43 milligrams.

Before operation temperature 97° to 99.9°, pulse 68 to 100, respirations not remarkable.

X-ray showed marked hypertrophic changes about the margins of the lumbar spine. There was calcification of the arteries of the pelvis. The left kidney outline did not appear abnormal. There were no visible stones. A film of the chest showed no abnormalities.

January 13 cystoscopy was done. The patient suffered a good deal. January 19 operation was done. After it the patient suffered much more. The inlying catheter became blocked, adding to his discomfort. He was heavily morphinized. January 23 the pain became much less severe. The drainage was from around the tube rather than through it. Beginning January 29 he had alternate diarrhea and constipation with pain in the rectum. February 3 he was irrational at times and the following day quite dull. The bowel condition improved, but the general condition became much worse. On and after February 7 the temperature was 96.9° to 100.1°, the pulse 79 to 120. He became irrational or stuporous most of the time. February 12 the urine showed a trace of albumin and was loaded with pus and débris. Morphia was used liberally. February 28 he died.

Discussion

BY HARVARD H. CRABTREE, M.D.

I am acquainted with the history and operative findings in this case, but not with the necropsy findings. The discussion is along the line of reasoning employed during his study and treatment.

This is a man of sixty-seven with a urinary history going back two years. Of course the first thought is of a possible prostatic situation. The story however is more of bloody urine and pain associated with it than of straining or retention or anything suggesting difficulty in passing urine. The absence of obstructive symptoms is against a prostatic enlargement. He might have bloody urine from enlarged prostate, particularly an ulcerated middle lobe. On the other hand if hematuria which began two years before had been due to ulceration from enlargement there would probably have been retention or threatened retention some time during those two years.

There was important time—three months—lost between the appearance of his symptoms and the date when diagnosis was seriously attempted. Then he was cystoscoped and three treatments were given, but the last was only three months before his admission to this hospital. If he had a neoplasm of his bladder the original cystoscopy certainly should have shown it and active treatment should have been instituted.

He had urinated once at night for ten years, suggesting an enlarged prostate, possibly. The frequency was increased to ten to twenty times at night and every half-hour by day just previous to admission. That of course might go with a bladder which was gradually filling up with residual, and the mention of dribbling would be consistent with that.

The rheumatism in his back is not surprising at his age; neither is dyspnea on moderate exertion.

The loss of ten to twenty pounds in a few months he might have had with chronic retention from prostate, or he might have had it from malignancy.

"Prostate diffusely enlarged and somewhat firmer than usual; marked tenderness." It is

difficult to say from this whether it was thought there was enough enlargement to cause retention or not. The tenderness is somewhat against an ordinary adenomatous prostate. There might well be tenderness with a malignant prostate or bladder base. If this prostate had been malignant I think it would have been described as hard or nodular,—at least not as "somewhat firmer than usual".

The urine, except for gross blood, does not help out in the diagnosis. A residual of only twenty cubic centimeters however is important evidence. Adenomatous enlargement of the prostate we can accept as pretty well ruled out. That is only one observation on the residual, and there is a possibility of error there. I mean that on one occasion he might empty his bladder better than on others, but I should not expect him to have as little as twenty cubic centimeters at any time if he had an obstructing prostate.

A renal function of sixty per cent. is very good. Again, a man with an obstruction going back two years would probably have a depressed function, a good deal below this.

The normal nitrogen is also against chronic obstruction with resulting kidney damage.

DR. GEORGE W. HOLMES: These spur-like projections on the margins of the spine we speak of as "hypertrophic spurs". They are about as common as arteriosclerosis. Whether they represent a true arthritis or not I do not know.

The arteries that we see usually are the ones rather deep in the pelvis, the internal iliacs. It would be impossible to demonstrate them in this light.

DR. CRABTREE: The X-ray evidence would lead us to expect that a neoplasm would be found rather than any particular enlargement of the prostate, and that the condition is not a neoplasm of the prostate but a neoplasm of the bladder.

PRELIMINARY DIAGNOSIS JANUARY 13

Carcinoma of the bladder.

CYSTOSCOPY

The cystoscope passed easily. A great deal of slough, mucus and pus was washed out of the bladder, but the median could not be got clear and the view obtained was imperfect for this reason and because of free oozing of blood. A mass of sloughing tissue was seen which appar-

The shadow of the injected bladder is not definitely abnormal in size but is abnormal in general outline. Occupying the greater part of the right half of the bladder and extending beyond the median line there is an area in which there is absence of the opaque solution. The area shows considerable variation in density and its margins are somewhat irregular. It might be produced by a large irregular growth in the bladder.

ently lay more to the right than to the left. The prostatic ring, at least anteriorly, looked normal.

CYSTOGRAM

The shadow of the injected bladder was not denitely abnormal in size but was abnormal in general outline. Occupying the greater part of the right half of the bladder and extending beyond the median line there was an area in which there was absence of the opaque solution. The area showed considerable variation in density and its margins were somewhat irregular. It might have been produced by a large irregular growth in the bladder.

FURTHER DISCUSSION

DR. HOLMES: The dark area on the plate of course represents the fact that Dr. Crabtree has just described. This represents the opaque mixture in the bladder. The bladder outline is often very irregular in normal people, and the rugae and spasm pressure, etc., make interpretation of these plates difficult. I think the man who does the cystoscopic examination is often in a better position to interpret these plates than we are. He knows what he is looking for and whether these appearances agree with what he sees. Such plates are however of use in determining the size of the bladder tumor even after cystoscopic examination has been done.

DR. CRABTREE: Would you have been willing to make a diagnosis of neoplasm of the bladder from X-ray alone?

DR. HOLMES: No. I should have said it showed a filling defect. Anything inside the bladder would produce such an effect. Occasionally we get mottling, finger-like markings around the tumor, which is quite suggestive of tumor.

DR. CABOT: Cancer was your diagnosis before operation?

DR. CRABTREE: Yes. The question might arise, after seeing such an extensive growth in the bladder and considering the X-ray and cystoscopic evidence, whether operation was justifiable. We undertook it with the idea that it was not going to be curative. The only thing, I think, which would justify it in this case would be the hope of relief of his pain by giving him permanent suprapubic drainage and stopping his bleeding. We did not expect that it would be found possible to remove a growth of this apparent size. The most that we hoped to do was to cauterize it or treat it with radium or perhaps do both, and drain his bladder.

PRE-OPERATIVE DIAGNOSIS JANUARY 19

Carcinoma of the bladder.

OPERATION

Gas-oxygen and anesthol. An extensive tumor involving the entire right side of the bladder was coagulated with a low diathermy current. Radium seeds were implanted in the tumor and around its edge and a suprapubic drain inserted.

PATHOLOGICAL REPORT

Two small fragments from the bladder showing on microscopic examination the structure of squamous cell carcinoma with considerable pearl formation. The histologic appearances suggest a growth of a relatively low grade of malignancy.

Squamous cell carcinoma.

FURTHER DISCUSSION

No attempt was made to remove it. He did fairly well for a while.

The mention of diarrhea and constipation and of pain in the rectum is interesting. I suppose a possible explanation of that would be the diathermy and possibly the radium. This tumor is in the right side, which would be a little away from the sigmoid. On the other hand it was so extensive and so low that it might have been directly over or beside the rectum, and the treatment have been the cause of the rectal symptoms which followed.

There is no mention of the relation of the ureter to this mass. It seemed to us at the time that it must necessarily have been involved, either going directly through the mass or so close to it that pressure on or actual invasion of the ureter might be expected to have some back pressure effect on the right kidney. His blood nitrogen and phthalein indicated a good total kidney function. On the other hand this mention of stupor, dullness, and irrational condition suggests that the man was getting uremic; his temperature of course was from sepsis and absorption. The X-ray does not show or state the size of the right kidney. Sometimes if the ureter is blocked the kidney seems to be increased in size. We have no evidence of that here. We quite expected, however, to find that that ureter was involved, that the right kidney was somewhat impaired and the man had been living more on his left one.

We thought that he died uremic and septic. A certain amount of sepsis would come from the slough and he would absorb, of course, a good deal from that and even if his kidneys were not already infected, he might easily get a secondary infection in one or both from that source. We thought this growth probably extended through the bladder wall and very likely was adherent to outside structures.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Carcinoma of the bladder.

DR. HARVARD H. CRABTREE'S DIAGNOSIS

Carcinoma of the bladder extending through the bladder wall and adherent to outside structures.

Probability of right ureteral obstruction.
Uremia.
Sepsis.

ANATOMIC DIAGNOSES

1. *Primary disease.*

Epidermoid carcinoma of the bladder.

2. *Secondary or terminal lesions.*

Chronic cystitis.
Chronic pyelonephritis.
Occlusion of right ureter.
Atrophy of right kidney.
Fibrous myocarditis.
Arteriosclerosis.
Lobar pneumonia.

Dr. Tracy B. Mallory: A large papillomatous cancer of the bladder was found occupying the right lateral, most of the anterior, and most of the posterior walls. Anteriorly it passed through the bladder wall and was already invading the periosteum of the posterior surface of the pubes. Posteriorly it had gone through the muscular wall of the bladder but had not as yet invaded the rectum. The right ureter was completely occluded by a tumor mass, and the right kidney, as one would expect, was atrophic, the right ureter dilated, and also the pelvis of the right kidney. There was practically no renal parenchyma on that side. The left kidney was markedly hypertrophied, suggesting that the closure of the ureter on the right side must have occurred a considerable time before to permit of compensatory hyperplasia on the opposite side. Many scars and minute purulent abscesses were found in the cortex of this last and relatively better kidney.

The immediate cause of death was a lobar pneumonia in his left lower lobe. There was a moderate degree of arteriosclerosis. The heart was negative, the other organs essentially negative.

Dr. Crabtree: Here was a man of cancer age, with progressing hematuria and bladder pain, allowed to bleed three months before he was cystoscoped and to waste twenty-one months more in half-hearted treatment. If hematuria were invariably regarded with the same seriousness as hemoptysis—and it obviously ought to be—then possibly his life, like many others, might have been saved.

The New England
Journal of Medicine

SUCCESSOR TO
THE BOSTON MEDICAL AND SURGICAL JOURNAL

Established in 1828

Published by THE MASSACHUSETTS MEDICAL SOCIETY under the jurisdiction of the

COMMITTEE ON PUBLICATIONS

HOMER GAGE, M.D. JOHN W. BARTOL, M.D.
ROGER I. LEE, M.D. ROBERT B. OSGOOD, M.D.
EDWARD W. TAYLOR, M.D.

EDITORIAL STAFF

DAVID L. EDSALL, M.D. STEPHEN RUSHMORE, M.D.
REID HUNT, M.D. HANS ZINSSER, M.D.
JOHN P. SUTHERLAND, M.D. BENJAMIN WHITE, PH.D.
GEORGE R. MINOT, M.D. HENRY R. VIETS, M.D.
FRANK H. LAHEY, M.D. ROBERT N. NYE, M.D.
SHIELDS WARREN, M.D.

WALTER P. BOWERS, M.D., *Managing Editor*

ASSOCIATE EDITORS

GEORGE G. SMITH, M.D. WILLIAM B. BREED, M.D.
JOSEPH GARLAND, M.D.

THE NEW HAMPSHIRE MEDICAL SOCIETY
PUBLICATION COMMITTEE

D. E. SULLIVAN, M.D. EMERY M. FITCH, M.D.
JOSEPH J. COBB, M.D.

THE VERMONT STATE MEDICAL SOCIETY
PUBLICATION COMMITTEE

WILLIAM G. RICKER, M.D. C. F. DALTON, M.D.
J. A. WARK, M.D.

SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, Canada $7.04 per year, $7.56 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 126 Massachusetts Avenue.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The New England Journal of Medicine, 126 Massachusetts Avenue, Boston, Mass.

CO-OPERATION

THERE has been so much disagreement, so much quibbling and so much petty jealousy between doctor and health agency that it is refreshing and inspiriting to find that New York State is in a fair way to find the solution which spells the arrival of the millenium when lion and lamb will lie down together. Which is the lion and which the lamb we will not dare to state; we humbly ask that a mere figure of speech will not be taken too literally. As in so many of the affairs of human life, friction in this case is caused, not by too close contact, but by too wide separation increasing the shock of occasional and unforeseen contact. Gears which mesh perfectly and are well lubricated mesh quietly and perform good service together; unadjusted gears clash noisily and serve only to injure each other.

Recognizing that coöperation and mutual understanding would do much to further the interests of all parties, leading members of the New York Medical Society, the State Charities Aid Association, Committee on Tuberculosis and Public Health, and the State Health Department met in November, 1925 in an informal conference to begin a discussion which might bring to light underlying conflicts. All phases of public health were discussed in detail and after their intricacies were thoroughly threshed out it developed that there was little, if any, real difference of opinion.

Two other similar meetings followed, and in the following November the State Committee on Tuberculosis and Public Health of the State Charities Aid Association adopted a resolution suggesting to the president of the State Medical Society the creation of a joint committee to carry on officially the work of coöperation that had been begun informally the preceding year. The preliminaries to the appointment of this committee and its final report were published in July 1927 by the State Charities Aid Association.

The report of this committee which dealt with general considerations as to relation between medical organizations and voluntary health agencies, was submitted to the House of Delegates of the State Society at its meeting in May, 1927, and was unanimously adopted. The Board of Managers of the State Charities Aid Association approved it later in the same month, and in the following month the State and Local Committees on Tuberculosis and Public Health of the S. C. A. A. unanimously approved it.

This report recognized that most of the basic principles on which public health methods are carried on were established by the wish of physicians, that the success of public health endeavors is directly proportionate to the interest taken in them by the medical profession, that the physician's activity in public health work should be encouraged and his coöperation solicited, and that the intimate relationship between physician and patient should be encouraged rather than disturbed.

Two sets of rules—of golden rules—were formulated by the Committee: one for the voluntary agencies, the other for the medical societies, and in these lies the kernel of the whole report; they are the hinges on which the door of mutual understanding swings.

SUGGESTIONS FOR VOLUNTARY AGENCIES

(a) Invite the County Medical Society to designate a certain number of its members, say two, to become members ex-officio of its board of directors or executive committee, if there be one.

(b) Lay its annual program of activities before the County Medical Society for information and suggestions.

(c) Appoint a special committee to confer with a similar special committee appointed by the County Medical Society about matters that

come up from time to time in which both groups are mutually interested.

(d) Report to the parent body of the voluntary health agency any matter as to which an accord has not been reached with the County Medical Society, in the hope that through conference between the parent body of the voluntary health agency and the State Medical Society any such matter may be negotiated and adjusted to the mutual satisfaction of all concerned.

(e) Conduct at least one open meeting each year to which the members of the County Medical Society are specially invited.

SUGGESTIONS FOR MEDICAL SOCIETIES

It is believed that these desirable results will also be furthered if the County Medical Society will include the following in its program:

(a) Accept the invitation from the voluntary health agency to designate certain of its members to serve as members ex-officio on the board of directors or executive committee of the voluntary health agency.

(b) Study and consider any program of activities that may be referred to it by the voluntary health agency for information and suggestions; and, if such program is approved, make a public statement to that effect, and otherwise support the program so approved.

(c) Appoint a special committee on public relations to confer from time to time with similar committees appointed by the voluntary health agency on matters of mutual interest to both agencies.

(d) Report to the State Medical Society any matter as to which an accord has not been reached with the voluntary health agency, in the hope that through conference between the State Medical Society and the parent body of the voluntary health agency any such matter may be negotiated and adjusted to the mutual satisfaction of all concerned.

We in Massachusetts hear a good deal of the type of back-biting and scandal-mongering that results from and begets mutual misunderstanding and dissatisfaction. The hospital interne cavils at the Social Service Department which is his best friend in helping him care for his patients; the officious social worker makes diagnoses and acts accordingly; the practitioner criticizes the hospital and the clinic for stealing his patients, and the Health Department for interfering with his practice.

A little understanding, a little coöperation, a little mutual consideration, and above all a little free discussion of the problem involved is all that is necessary to bring about a cordial relationship. Cannot we, also, organize in the larger sense for the benefit of humanity?

THE FILTERABLE VIRUSES

THERE are few more fascinating fields in medicine today than the study of that wide group of diseases caused by the so-called "filterable viruses", substances whose nature is in dispute, and a number of which apparently come from the unknown and depart into the unknown, leaving only disease or death as token of their passage. There is no doubt but that included in this group of diseases due to the filterable viruses are a number of diverse conditions. Inasmuch as an arbitrary unit of size is the only standard for the admission of disease-producing agents into this group, we know certain of them to be minute bacteria, others we know to be living substances, and still others are as yet in question as to whether they are animate or inanimate. Any number of lesions are traced to agents apparently belonging in this group, ranging from infectious chloroses of plants, jaundice of silk worms, hog cholera, lymphocytic disease of fish, to such every day diseases as warts, measles, and vaccinia.

The importance of some of these diseases, such as encephalitis and poliomyelitis, has only been recognized in recent years. As is pointed out by Rivers[1], the group is no more homogeneous in regard to its epidemiology than in any other aspect, ranging from the extremely contagious diseases such as smallpox, to the Rous sarcoma of chickens, transmitted only by inoculation.

Certain of the viruses, in addition to the specific character of the disease which they produce, cause definite intracellular changes, some of which have long been familiar, such as the Negri bodies in rabies. It is to be hoped that through the study of these viruses in their hosts or in cultures of tissues from hosts that it will be possible to throw new light on the problems that confront us in their control. The fine example that Pasteur has given us of the ability to master one of the most important of the diseases due to filterable viruses even though ignorant of its exact nature, through his discovery of the mode of transmission and the prophylactic inoculation against rabies, should lead to conquests of other of these little understood diseases, perhaps even without waiting for the essential but slow and blind groping toward complete understanding of them.

REFERENCE

1 Rivers, Thomas M.: Filterable Viruses. Williams & Wilkins Co., 1928.

SOME DEFICIENCES IN DEALING WITH TUBERCULOSIS

FOR more than a generation Massachusetts has given cencentrated attention to the problem of tuberculosis. This state has been in the front line of attack on this disease. It has built and caused to be built sanatoria for the reception of tuberculosis patients. Its Department of Public

Health has done excellent work through State institutions and campaigns of education. Voluntary local and state organizations have coöperated with official health bodies.

The Massachusetts Medical Society has a section devoted to tuberculosis which places before the profession during the Annual Meetings the most important features incident to dealing with this disease.

The main purposes of the combined efforts of public and voluntary tuberculosis organizations are first prevention through the promotion of better health, second early diagnosis of the disease and third treatment.

The field of prevention has been well cultivated and reasonable efficiency seems to have been established. Unfortunately early diagnosis of the disease is not as general as it should be.

This defect in the attack on the disease can be laid to the practitioner in too many cases although the indifference of the patient is a contributing factor. It may be too much to expect that there will be general recognition of the pre-tuberculosis conditions where there may be so slight evidences of latent infection that only the expert can be sure of the diagnosis, but it is fair to demand of the practitioner that all cases of lowered vitality which are not due to some obvious cause should be studied to establish or rule out the diagnosis of tuberculosis. If the easily demonstrable lesions in the lungs, however, are not detected by the family physician, the question may be raised with respect to the competency of such doctors. It may not be a stretch of the imagination to conceive of official action when such lapses are found to have existed.

The recent report of a case to the Commissioner of Public Health of Massachusetts is pertinent and reads as follows:

I visited Mary D. at 10 A. M. Wednesday, April 11th. This is the story: In June 1927 this girl first felt ill and sought the advice of her family physician who gave her a tonic; said nothing about discontinuing her work. She continued to work for about six weeks, growing steadily worse and believing that the "tonic" which the doctor gave her made her worse instead of better. One day she fainted, returned to her doctor and asked him if he thought there was something wrong with her lungs, as she had developed a cough, which was dry and persistent. After examining her he said "No, there is nothing wrong with your lungs and don't let anyone tell you that there is." Gradually feeling worse she stopped work herself and thought that going to the country would help her. She went in July to New Hampshire and there met a nurse who advised her to go and see an expert.

This she did in October and he, after examining her said she was a proper candidate for Rutland.

This illustrates a defect in medical practice the record of which is in the possession of the State Department of Public Health.

The examining physician recommended this patient for admission to the Rutland Sanatorium but the waiting list then on file prevented her entrance.

It is true that a bed was available at Mattapan but the patient and her friends preferred to wait for an opening at Rutland.

This opens the question long a source of worry to our health authorities with respect to the responsibilities of the State.

If doctors recommend patients to certain sanatoria the patient may not be willing to accept a substitute.

With the recognized advantage of sanatorium care this case (which is not unique) raises the question as to the responsibilities of Health Authorities. The addition to the number of sanatoria as required by the passage of a bill by the Legislature recently, requiring two country institutions to be built may help the situation as it applies to sanatoria.

Further attention to early diagnosis is apparently a very important field for concentrated propaganda.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

MILLER, RICHARD H. A.B., M.D. Harvard, 1910. F.A.C.S. Assistant Visiting Surgeon, Massachusetts General Hospital; Assistant Professor of Surgery, Harvard Medical School. Address: 270 Commonwealth Avenue, Boston. Associated with him is:

ROGERS, HORATIO. A.B., M.D. Harvard, 1923. Assistant in Surgery, Massachusetts General Hospital. Address: 270 Commonwealth Avenue, Boston. Their subject is: "Sarcoma of the Uterus". Page 927.

FINE, JACOB. A.B., M.D. Harvard, 1924. Formerly Assistant Resident in Surgery, Massachusetts General Hospital; Interne at Lakeside Hospital in Cleveland; Graduate Assistant at Massachusetts General Hospital. Address: Nantucket, Mass. Associated with him is:

BROWN, SAMUEL. S.B., M.D. Harvard, 1927. Formerly Assistant Resident Medical Officer at Boston Sanatorium. Address: 672 Second Street, Fall River, Mass. Their subject is: "The Influence of Parathormone on Bone Regeneration". Page 932.

TERHUNE, WILLIAM B. M.D. Tulane School of Medicine, 1915. Austen Riggs Foundation. His subject is: "Psychopathology and Treatment of the Psychoneuroses". Page 937. Address: Stockbridge, Mass.

EINHORN, MOSES. Herzlia College, Tell Aviv Palestine, M.D., University Bellevue Medical College, 1922. For 4 years on the Gastro Enter-

ological Staff of the Outpatient Department of Mount Sinai Hospital; Gastro Enterology, Beth Israel Hospital. Inventor of several gastroenterological instruments. His subject is: "Minim Method for Analysis of Gastric Content". Page 941. Address: 983 Park Avenue, New York City.

JORESS, MARK H. M.D. Tufts, 1920. Resident Physician, Rutland State Sanatorium, 1922-1927; Examining Physician, Boston Health Department; Division of Tuberculosis, 1927-1928; Teaching Assistant, Department Pulmonary Diseases and Climatology, Tufts Medical School, 1927-1928. His subject is: "Differential Diagnosis in Pulmonary Diseases". Page 943. Address: 483 Beacon Street, Boston.

CLIFFORD, RANDALL. A.B., M.D. Harvard, 1917. Assistant in Medicine, Harvard Medical School; Physician in Charge of Pulmonary Clinic, Massachusetts General Hospital; Consulting Physician, Massachusetts Eye & Ear Infirmary; Visiting Physician, Channing Home; Secretary of Trudeau Society of Boston. His subject is: "The Interpretation of Apical Râles In Pulmonary Tuberculosis". Page 948. Address: 475 Commonwealth Avenue, Boston.

NELBACH, GEORGE J. B.A., B.L. Cornell, 1907. Executive Secretary, State Committee on Tuberculosis and Public Health, New York State Charities Aid Association, from 1911 to date; Member, Board of Directors, National Tuberculosis Association, May 1927-May 1929; President, National Conference of Tuberculosis Secretaries, 1915. His subject is: "Fundamentals in Tuberculosis Work with Respect to Voluntary Unofficial Association". Page 952. Address: 105 E. 22nd Street, New York City.

THE ANNUAL MEETING OF THE AMERICAN MEDICAL ASSOCIATION

THE American Medical Association was the guest of Minneapolis, June 11 to 15, 1928, for its annual convocation.

This city is one of the more progressive municipalities of the middle west and played the part of host to the largest medical society of the world most acceptably.

Banners, flags and placards of welcome were in general evidence.

The accommodations in the auditorium for the exhibits and the Annual Meeting were ample.

The commercial exhibits numbering about one hundred and seventy were well displayed and covered the range of useful and necessary adjuncts to medical art. A visitor might be led to wonder whether humanity need ever be ill with all the foods and medicines displayed and recommended to meet all of the deficiencies of nutrition and the inroads of disease. Even our over-enthusiastic California Fruit Growers Association was on hand to cure the eighty or more million of the people of this country of the claimed well-nigh universal acidosis which in the economic workings of nature may have been imposed on the race in order to promote the prosperity of that altruistic organization.

The multitude of surgical instruments and appliances is convincing evidence of the ingenuity of the inventive mind and together with the various electrical and light giving mechanisms suggests that if one could equip a sufficiently extensive suite of rooms with the instruments which the vendors would impose on the doctor the practice of medicine would be an exact science provided one had the capacity to understand and apply all of the devices recommended, and absorb all of the available literature.

The scientific and educational features of the exhibits were of commanding interest. Almost all departments of pathology were well represented and compilations of statistical facts relating to medical education, the distribution of physicians and the work of examining boards were well displayed, making an excellent postgraduate course.

The sections were well attended and the papers and discussions were especially interesting.

A great deal of interest was shown in the work done in Boston, especially that of Minot and Murphy.

Massachusetts men were much in evidence in the programs and audiences.

The Annual Meeting Tuesday night was attended by more than two thousand Fellows and guests of the Association, and the auditoriums provided ample accommodations for the large audience.

Dr. William S. Thayer's Presidential address was a carefully prepared resumé of the problems of especial interest to the profession with well chosen advice to maintain the best traditions of medicine and especially exert all possible influence in promoting public health organizations and measures.

He felt that all proper influence should be exerted by the profession to bring about the selection of well qualified members of public health bodies.

Governor Christianson welcomed the Association to the State in an address which showed that he is in cordial sympathy with the medical profession and pledged himself to give all possible support to all efforts designed to ameliorate suffering and prevent disease.

Governor Christianson is an orator of high rank and his address was an especial feature of the occasion.

As a convention city Minneapolis ranks high both because of ample accommodations and its spirit of hospitality.

In the closing session of The House of Delegates, Dr. M. L. Harris of Chicago was elected President Elect to take office one year hence, and Portland, Oregon, won the honor of the place for the 1929 meeting.

Dr. Fred B. Lund continues as a member of the Committee on Medical Education. Dr. Charles E. Mongan of Somerville continues as a member of the Committee on Reports of the Trustees to the House of Delegates. Massachusetts is also honored by the appointment of Dr. Reginald Fitz of Boston as a member of the Committee to take the place of Dr. Bevan, who has held this position for many years.

The *Bulletin* recorded members from the New England States as follows: Maine 6, New Hampshire 7, Vermont 5, Rhode Island 5, Connecticut 15, and Massachusetts 67. The total registration reported on Friday, the last day, was 4802.

THE MEETING OF THE HARVARD MEDICAL SCHOOL ALUMNI IN MINNEAPOLIS

As a feature of the meetings of the American Medical Association many organizations and groups appreciate the opportunity to meet and renew acquaintances of former years and compare notes.

One of the enjoyable occasions at this year's meeting was a dinner of the graduates of the Harvard Medical School who were attending the meetings of the A. M. A. at the Woman's Club of Minneapolis on the evening of June 13. Dr. P. E. Truesdale of Fall River, the President of the Alumni Association, presided and called upon many of the Alumni for after-dinner remarks. He kept the sustained interest of the Alumni by apt illustrations and appropriate references to facts connected with the development of the School in recent years, between the speeches of those who responded to him.

Dr. Elliott P. Joslin, the first speaker, conveyed to his audience his unbounded enthusiasm in all matters connected with the school. He told of the response of the Alumni to the appeals for contributions to the building fund for the dormitory, explaining that the general response was one important factor in leading Mr. Vanderbilt to make his generous donation without which the building would have had to be deferred for a long time.

He related several delightful experiences in connection with his visit to Minneapolis in visiting former students of the Medical School, and referred especially to the diagnosis of diabetic coma in a child ill with mastoid disease by a graduate of the School. This Dr. Joslin felt was a tribute to the acumen of the doctor and a credit to the School. In a delightfully cordial way he called upon many of the men who are successful in their various fields to stand as he introduced them as examples of the finished product of the School.

He paid tribute to the scientific work in Minneapolis, a notable example being shown by the record of fifteen autopsies in every one hundred deaths in this city.

Dr. Roger I. Lee found in his association with Dr. F. C. Shattuck the inspiration for his remarks and told story after story setting forth Dr. Shattuck's influence as a teacher on the students, the brilliant and original manner of conveying his thoughts and his experiences with his associates in the everyday affairs of life.

Dr. Lee's remarks were especially enjoyed because of the great popularity of Dr. Shattuck who was not only a successful teacher but one whose interest in the students was so keen that all came to regard him as a personal friend.

Dr. Frank H. Lahey reported the experience of a visitor to the clinic of a popular surgeon when he heard the recital of a particular formula, four questions and answers which the staff had to learn in order to have impressed upon their minds the good fortune enjoyed by those who were privileged to be in that service. This surgeon, however, is not a graduate of the Harvard School and the illustration may have been intended to show the differences in the advertising methods of some as compared with the modest conservatism of Harvard men. Dr. George H. Bigelow responded to the suggestion that he is the leader in public health matters in Massachusetts and like a Gatling gun let loose a rapid fire monologue of pungent stories in his unapproachable style. We venture to assert that, of all graduates, his mental machinery is geared to the highest possible speed and that his illustrations are picturesque to a superlative degree. He won hearty applause.

A reference to the development of the NEW ENGLAND JOURNAL OF MEDICINE was made by Dr. W. P. Bowers with the explanation that the JOURNAL has been kept alive by Harvard men in the past. He also took the opportunity to pay tribute to Dr. F. C. Shattuck, who supported the JOURNAL liberally, and to his brother, Dr. George B. Shattuck, who served as its Editor for many years. Dr. William Robey expressed pleasure in meeting men who had been his students in the School and the Boston City Hospital and called to mind some of the salient characteristics of the staff of the institution which were evidently appreciated by many of those who had served as internes in the hospital. Dr. Fred H. Albee of New York expressed his pleasure in having been led to choose Harvard rather than some other medical school and told of the engrossing requirements of his work as prosector in the school which had prevented his acceptance of the invitation to play football on the Harvard team. Dr. J. S. Stone was asked to speak on a new departure in the policies of the Massachusetts Medical Society but, since this departure applied to certain functions imposed on him, he simply referred to the position of Executive Secretary as an experiment still in the trial stage. He did, however, find in his association with some of the prominent surgeons connected with the school certain occurrences which furnished material for interesting recollections. Dr. Ed-

mond F. Cody of New Bedford and Dr. Roland Hammond of Providence, both members of the House of Delegates of the A. M. A., explained the functions of this body and testified to the importance of the work done in the House and referred to the great influence which is being exerted by the Association in the elimination of sub-standard medical schools and quackery. Dr. David Parker of Manchester, N. H., brought the congratulations and good wishes of his state to Minnesota speaking of both states as having distinctive charms which make them attractive. He expressed approval of reunions and his appreciation of the standing of the Harvard Medical School and pride in the achievements of its graduates.

Dr. Sidney Storrs Hall of Minneapolis was presented as the oldest representative present, having graduated in 1867. He compared the education of his day with that of the present and felt proud of the contributions to medicine by the Alumni. All rose in recognition of the long service to humanity by this still young appearing veteran in medical ranks.

The last speaker was Dr. Archie H. Beard of Minneapolis to whom belongs the credit of having arranged the details of the alumni gathering. He gave an interesting recital of some of the details of his work and expressed his pleasure in the hearty response to the effort to bring the alumni together. He was given hearty thanks for his success in the perfect details of the meeting.

That the meeting was unusually successful was shown by the sustained interest up to midnight.

The list of men who attended follows:

W. R. Ohler, 1914, Boston, Mass.
Nathaniel Allison, 1901, Boston, Mass.
Fred H. Albee, 1903, New York, N. Y.
James S. Stone, 1894, Boston, Mass.
C. Guy Lane, 1908, Boston, Mass.
Samuel Ayres, Jr., 1919, Los Angeles, Calif.
Charles L. Martin, 1919, Dallas, Texas.
Monroe A. McIver, 1917, Boston, Mass.
Eric Stone, 1918, Providence, R. I.
E. K. Steenburg, 1918, Aurora, Neb.
Theodore H. Aschman, 1917, Kansas City, Mo.
Philip A. Kober, 1909, Evanston, Ill.
Lawrence W. Smith, 1920, Boston, Mass.
George H. Bigelow, 1916, Boston, Mass.
Frank Conger Smith, New York University Medical College, 1894, Yankton, S. D.
E. Stanley Abbot, 1893, Boston, Mass.
George H. Thomas, 1895, Minneapolis, Minn.
Frank R. Ober, Tufts College Medical School, 1905, Member of Harvard Medical School Faculty, Boston, Mass.
Arthur T. Mann, 1896, Minneapolis, Minn.
Archie H. Beard, 1914, Minneapolis, Minn.
Norman P. Johnson, 1923, Minneapolis, Minn.
Edward T. Evans, 1922, Minneapolis, Minn.
John F. Curtin, 1915, Minneapolis, Minn.
David W. Parker, 1903, Manchester, N. H.
John H. Morse, 1901, Minneapolis, Minn.
Edwin W. Ryerson, 1897, Chicago, Ill.
F. C. Kidner, 1904, Detroit, Mich.
Raymond J. Reitzel, 1924, Galveston, Texas.
Joseph P. Evans, 1929, La Crosse, Wis.
Warner Ogden, 1920, St. Paul, Minn.
Harry Oerting, 1917, St. Paul, Minn.
James A. Evans, 1920, La Crosse, Wis.
Hilding Berglund, Medico-Chirurgical Institute, Stockholm, 1916, Minneapolis, Minn.
William H. Robey, 1895, Boston, Mass.
E. P. Joslin, 1895, Boston, Mass.
Roger I. Lee, 1905, Boston, Mass.
Roland Hammond, 1902, Providence, R. I.
Frank H. Lahey, 1904, Boston, Mass.
Edmond F. Cody, 1893, New Bedford, Mass.
Sidney Storrs Hall, 1867, Minneapolis, Minn.
Arthur T. Legg, 1900, Boston, Mass.
Paul Starr, Rush Medical College, 1922, Chicago.
Elmer H. Heath, 1923, Baltimore, Md.
Theodore K. Selkirk, 1923, Cincinnati, O.
J. Victor Greenebaum, 1911, Cincinnati, O.
Lewis M. Hurxthal, 1923, Boston, Mass.
Derrick T. Vail, Jr., 1923, Cincinnati, O.
Franc D. Ingraham, 1925, Boston, Mass.
Kenneth L. Dole, 1915, Redlands, Cal.
O. H. Meuller, 1910, Los Angeles, Cal.
J. Archer O'Reilly, 1906, St. Louis, Mo.
J. S. Kahn, 1904, San Antonio, Tex.
S. Judd Beach, 1905, Portland, Maine.
R. Russell Best, 1922, Omaha, Neb.
Everett E. Angle, 1921, Lincoln, Neb.
H. E. Chamberlain, 1921, Minneapolis, Minn.
Horatio Sweetser, Jr., 1921, Minneapolis, Minn.
Philemon E. Truesdale, 1898, Fall River, Mass.
Walter P. Bowers, 1879, Clinton, Mass.

Several other graduates of the School were in the city, but were unable to attend because of other engagements.

BOSTON MEDICAL LIBRARY

Extension Courses for Rural Practitioners

The problem of keeping abreast of the progress of medical science is a serious one. To do so implies that the busy practitioner takes two or three of the best journals and reads them seriously, attends medical society meetings in his own locality and goes to one or two of the larger State or National Annual Conventions, where through intercourse with his confreres and by listening to papers he learns of what is going on. To do this in the midst of the exacting calls of a general practice is more than all but a very small minority of men will undertake. Attending meetings takes time that many feel they can ill afford. Reading journals at the end of a fatiguing day is a profitless endeavor which, by the majority, is soon abandoned. Post-graduate courses cannot be looked upon as a substitute for a vacation. The agents of drug houses and the purveyors of physiotherapeutic appliances bring about the only contacts with the outside world of medicine which many practitioners suffer themselves to enjoy. If there is a local hospital there is in it, sometimes, an incentive as well as an opportunity to bring oneself a little more in touch with what is going on. Borrowing from the University extension movement, which has been so successful in meeting the wants of individuals in academic fields, there have been quite a few

attempts to carry this idea into medicine. Geographical considerations determine to some extent the way in which this is undertaken. North Carolina, Pennsylvania, Wisconsin and Michigan, to single out a few where a scheme of this sort has been carried out, have been very successful in their endeavors. New England presents many of the same problems that the States above mentioned do, so far as the isolation of rural practitioners is concerned, and there is a serious dearth of young physicians in these communities. Neither is it at all likely that efforts to lure them into the country will prove successful until they see the opportunity to secure some of the post-graduate advantages which are enjoyed by those who have taken up their practice in the larger centers of population. Again New England is suffering, because two of its medical schools have been forced out of existence, schools where its native sons were willing and could afford to go and from which upon graduation they were far more likely to return to their own homes than has been the case with the comparatively few who could get together the means to take them to distant cities. The widespread distribution of the small cottage hospital throughout New England, would make the problem of carrying extension courses in medicine to these hospitals, which are frequently located in small towns providing service to a considerable territory, a comparatively easy one. Instead of the local doctors having to take time off for occasional post-graduate training in a remote city, courses could be brought to the practitioner with such frequency as his appetite for experiences of this sort seemed to demand. The expense would be less than going to the city and all the physicians in the section could be accommodated at once. Furthermore the distracting features which characterize the big meetings would be removed and more good, proportioned to the unit of time spent, would be obtained than from the large meetings which have taken on the characteristics of a junket for many who attend. These small community hospitals could be made the repositories of the sort of medical literature they need, drawn from the large metropolitan medical libraries. If this were done and periodic extension clinics held in these hospitals and centers, a taste for medical literature and a desire for progress would be cultivated and it would soon be recognized that the time spent in taking advantage of such opportunities would be well spent. This is something to the accomplishment of which the New England Medical Council might well devote some of its energies. The level of practice would be raised where it needs it so that the public would benefit; the profession would be stimulated to better work; the medical schools would doubtless indirectly profit by securing some matriculates of the right sort from rural communities, the hospitals would give better service, for the reason that making them a base for instruction invariably has that effect, and it may be that Dartmouth and Bowdoin would reëstablish their medical departments on a four-year basis and help to solve the problems growing out of the scarcity of general practitioners in New England.

For those who recognize some of these problems as existing for New England and who are interested in their solution the Library will put out the literature on certain of the extension-service schemes that have been in operation in other localities. This will be done during the week of June 24th.

MISCELLANY

NORFOLK COUNTY HEALTH ASSOCIATION STUDY

The Norfolk County Health Association, Inc., has just completed a study of 205 children who were sent to the Norfolk County Health Camp, St. Augustine's Farm to the Sharon Preventorium between the years 1924 and 1927. In this study, 22 of the 28 towns in the County have been represented.

These children were referred to the Camp through the Ten Year Program Clinic of the State Department of Health, dispensaries, school physicians, private physicians and other sources because of the need of supervision for the youngsters. Out of this group, 67 were referred for suspicious tuberculosis, 82 contact tuberculosis, and 56 because they were 10% plus underweight. Most of the children were required to go to Camp only once. Only 31 were sent twice, 12 three times and 5 four times. Investigation showed that 10 came from very good home surroundings, 34 from good, 61 from fair, 24 from poor, 15 from bad and 61 cases from unknown surroundings. A check of these home conditions in 1928 showed no material change.

At present there are 193 of these children still living in the County, of the other twelve, three are dead from accident and nine have left Norfolk County. In cases where the children have moved, care has been taken to notify the proper authorities so that adequate supervision may be maintained. Of the 193, ninety-seven have gained, thirty no gain or loss and nine are in hospitals or preventoria.

Much praise is due to the Norfolk County Health Association, Inc., which has made remarkable progress in the work for prevention of tuberculosis and represents one of the leading counties in the State striving for its control and elimination.

NEW YORK MEDICAL CENTER

The New York Medical Center continues its rapid advance toward completion. Starting on June 4 the Sloane Hospital for Women began to be moved to the new center; a process that consumed about two weeks. The Presbyterian Hospital at the Center is already operating to capacity, and the College of Physicians and Surgeons is partially functioning. The Squier Urological Clinic is in operation, and the Presbyterian Hospital School of Nursing now has 152 student nurses, all living in the Anna C. Maxwell Hall. The Vanderbilt Clinic will move to the Center on or about July 1, the New York Psychiatric Institute and the School of Dental and Oral Surgery of Columbia University in the fall, and the

Neurological Institute about the first of January, 1929. No exact date has as yet been set for completion of the Babies Hospital new building.

CORRESPONDENCE

OFFICIAL ACTIONS BY THE BOARD OF REGISTRATION IN MEDICINE

The results of the Board meeting of June 7th are as follows:

Dr. James H. Morris, of New Bedford,—laid on table until next meeting.

Dr. F. W. Doyle, of Northampton,—suspended for one month on charge of gross mis-conduct as shown by revocation of his prohibition permit.

Dr. Peter Kane,—registration suspended for three months on the charge of gross mis-conduct in the practice of his profession; and the registration of

Dr. Francis F. Henderson was suspended for one month on the ground of gross mis-conduct in the practice of his profession.

FRANK M. VAUGHAN, *Secretary.*

NOTICE

Remember to file Narcotic Registration papers before July 1.

REPORT OF MEETING

THE LAWRENCE MEDICAL CLUB

The monthly meeting of the Club was held Monday evening, May 28, with Roy V. Baketel, M.D., 302 Broadway, Methuen. The Chairman for the evening was J. Forrest Burnham, M.D. A paper entitled "Advances in Diagnosis and Treatment of Heart Disease" was read by Channing Frothingham, M.D., Boston.

The Chairman described the plan which the Massachusetts Medical Society is putting into operation, by which, through individual subscriptions, from Fellows, an amount of $125,000 is desired with which to purchase a headquarters for the parent Society. Essex North District Medical Society has appointed a committee of 32, each one of which will interview six members before July 15, 1928. The money will be sent to A. K. Stone, M.D., Treasurer of the State Society, Auburn Street, Framingham Centre, Massachusetts for the Building Fund. With a Fellowship of 4422 in the State Society, an average subscription of $31.00 will raise the required amount.

The Lawrence Medical Club, at this meeting, endorsed the morbidity survey which is now being made in Lawrence, June 4 to 30 inclusive, by the State Department of Health.

SOCIETY MEETINGS

June 21—Union Hospital in Fall River. Detailed notice appears on page 926, issue of June 14.

December 3-7—Radiological Society Convention. Detailed notice appears on page 712, issue of May 17.

BOOK REVIEWS

Filterable Viruses. Edited by THOMAS M. RIVERS. Williams & Wilkins Company. 1928. Pp. 428.

This volume deals with the general aspects of the filterable viruses and detailed studies of certain members of the group that have attracted attention recently. One of the most valuable chapters from the point of view of the laboratory worker is that by Mudd on filters and filtration. There are few authoritative articles on the various types of filters and their peculiarities. In this section by Mudd is a useful and comprehensive summary of our present knowledge.

The chapter dealing with intercellular pathology in virus diseases is also of considerable interest, particularly to the cytologist. Amoss contributes a valuable review of poliomyelitis. The bacteriophage is also included in this group of diseases and the section on this subject, written by Bronfenbrenner, is highly stimulating.

A valuable feature of the book is a carefully prepared bibliography, and in addition there are a number of excellent illustrations.

The Ultra-Violet Rays. By ARNOLD LORAND, M.D. (Vienna). Published by F. A. Davis Company, Philadelphia, 1928.

This book may have been written for laymen rather than for physicians. It presents a rather general and at the same time a rather superficial discussion of ultra-violet therapy. For the laymen a certain amount of interest and enthusiasm might be aroused, but for the physician and other scientific minded individuals there is little of value. Even on the subjects on which the author lays most stress, namely, preventing the change in color of hair and preventing loss of hair, one gets the feeling that he has not proved his points, even though the rays are useful in certain scalp conditions. There is considerable repetition, there is too much vagueness, there is too much reference to endocrine glands, and to the sympathetic nervous system, and nervous conditions in general, to make the book one of real value to the medical man.

An Elementary Text-Book of General Microbiology. By WARD GILTNER. P. Blakiston's Son & Co., Philadelphia, 1928.

This book contains a surprising amount of information about microörganisms. Their morphology, culture and physiology are well described, there are excellent chapters on applied microbiology, as well as on microbial diseases of man, other animals and plants. The material has been carefully chosen with a view to general acceptance and the purpose of the book, and the errors are few. The consistent use of the newer classification and terminology will be of help to older bacteriologists and other microbiologists. One might question the author's sense of fitness in using on page 212 cuts of pasteurizing plants that are so favorable to the firm supplying them. Their omission, the deletion of the many "verys" in the text, a better definition for epidemic on page 266, and the amplification of the chapters on the structure of bacteria, and their chemical composition, and of that on enzymes would leave the reviewer with only complimentary things to say of this useful and interesting text-book.